



ENVIRONMENTAL AND OCCUPATIONAL INTERSTITIAL LUNG DISEASES



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- pneumoconiosis
- drug-induced ILD
- HP

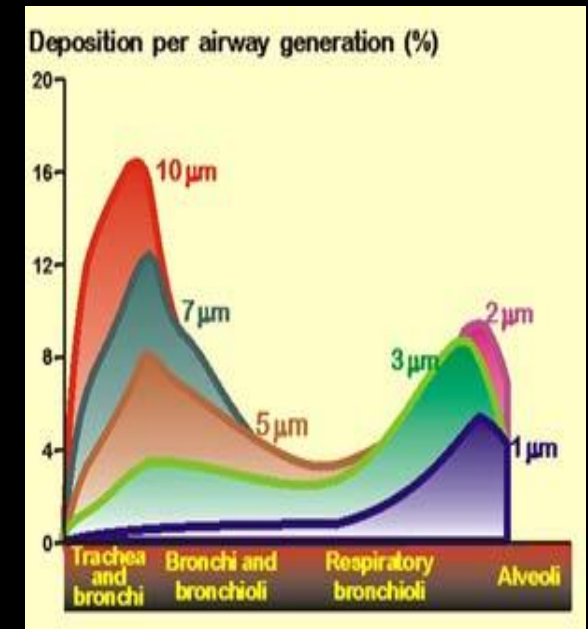
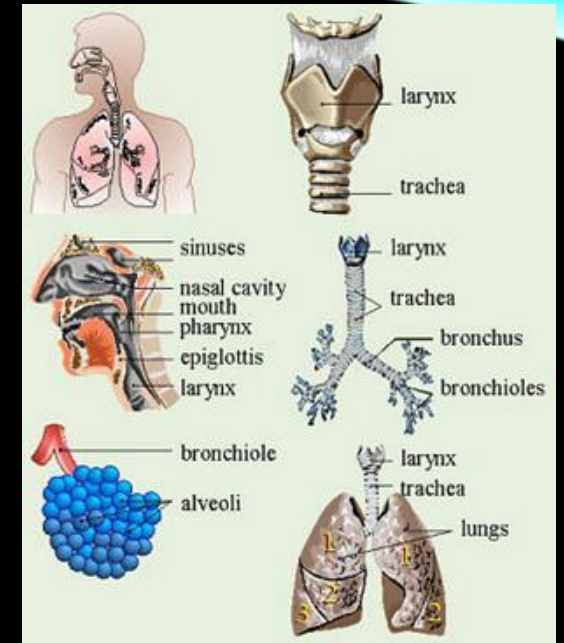


ENVIRONMENTAL AND OCCUPATIONAL INTERSTITIAL LUNG DISEASES

- Pneumoconiosis inhalation of mineral dusts
 - silica
 - coal dust
 - Asbestos
- HP inhalation of organic dusts

Fate of inhaled particles

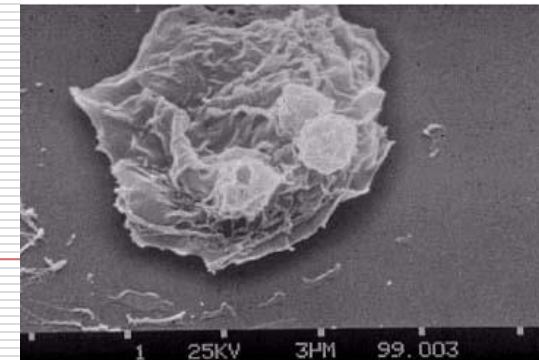
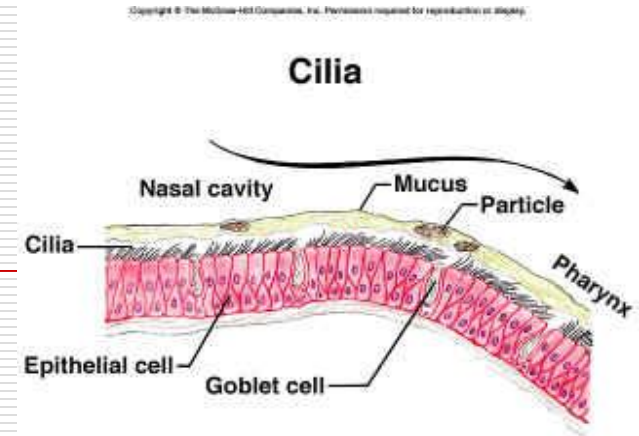
- *Large particles may get trapped in the nose or large airways,*
- *but very small ones may reach the lungs.*
- *There, some particles dissolve and may be absorbed into the bloodstream;*
- *most solid particles that do not dissolve are removed by the body's defenses .*



Body's defense mechanism

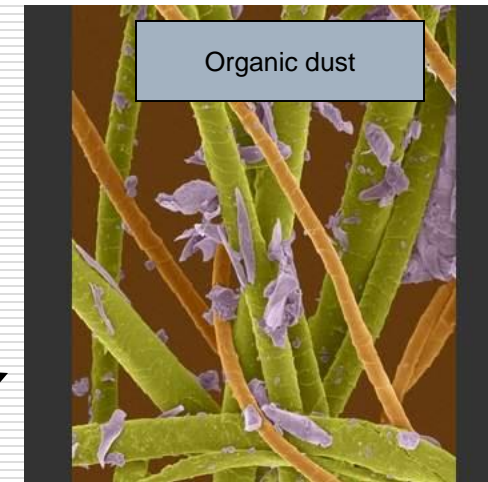
- *The body has several means of getting rid of inhaled particles.*
- *In the airways, an accumulation of secretions (mucus) coats particles so that they can be **coughed up** more easily.*
- *Additionally, tiny cells lining the airways (cilia) are able to **brush inhaled particles upward**, out of the lungs.*
- *In the small air sacs of the lungs (alveoli), special scavenger cells (macrophages) **engulf most particles and render them harmless.***

Phagocytosis



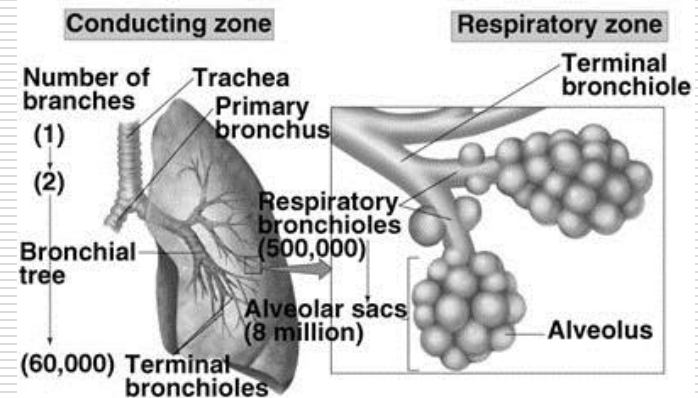
Nature of inhaled particles

- *Many different kinds of particles can harm the lungs.*
- *Some are **organic**,*
 - *such as grain dusts, cotton dust, or animal dander.*
- *Some are **inorganic**,*
 - *such as asbestos, and silica.*



Effects of inhaled particles

- *Different types of particles produce different reactions in the body.*
- *Some particles can cause **allergic reactions**, such as hay fever-like symptoms or **a type of asthma**.*
- *Other particles cause harm not by triggering allergic reactions but by being **toxic to the cells** of the airways and air sacs in the lung.*

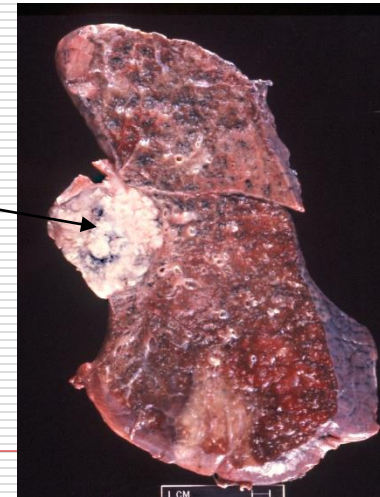


Effects of inhaled particles

- Some particles, such as *quartz dust and asbestos*, may cause *chronic irritation* that can lead to scarring of lung tissue (*pulmonary fibrosis*).

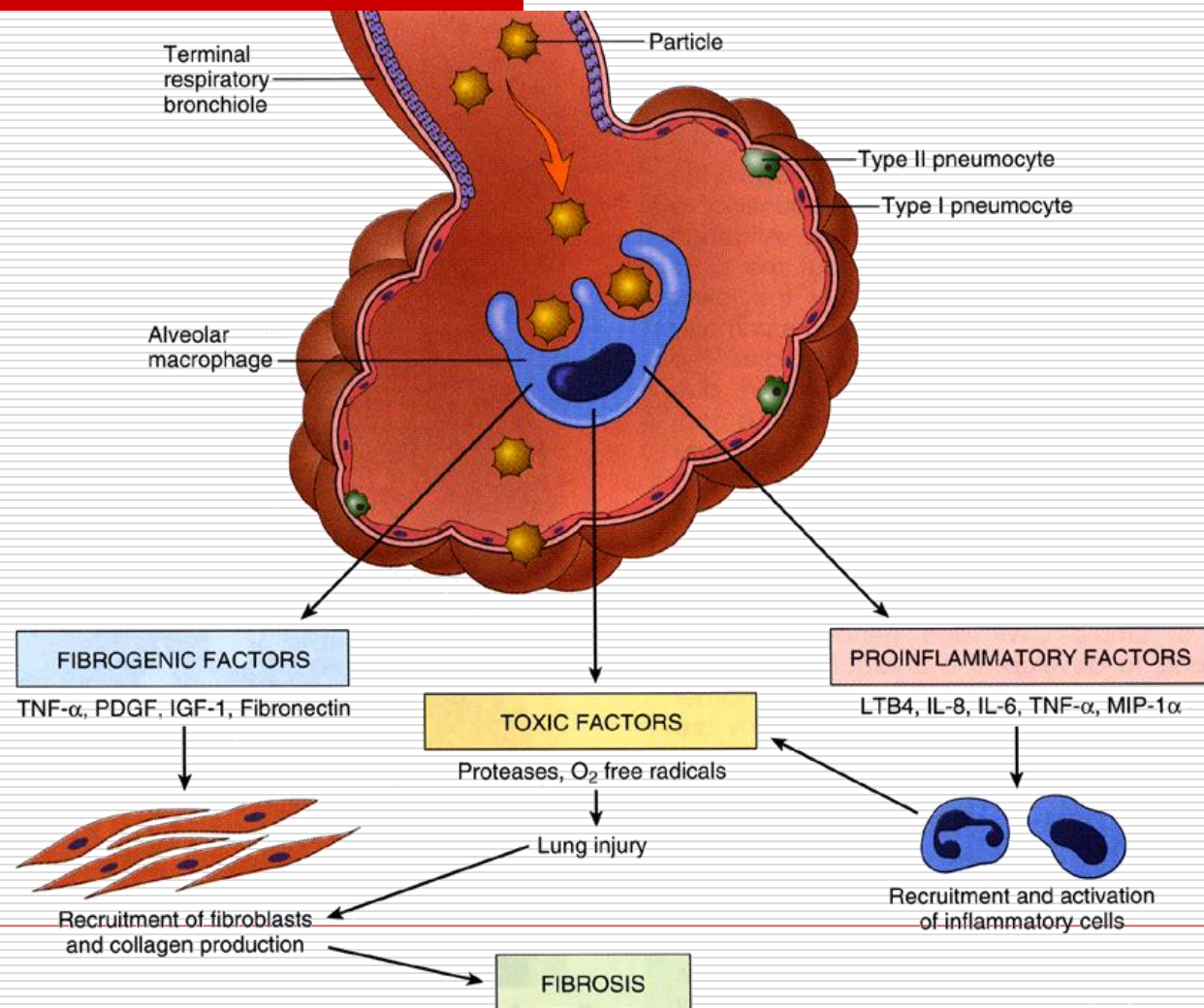


- Certain toxic particles, such as asbestos, can cause *lung cancer*, especially in smokers, or cancer of the lining of the chest and lung (mesothelioma), regardless of the person's smoking history.

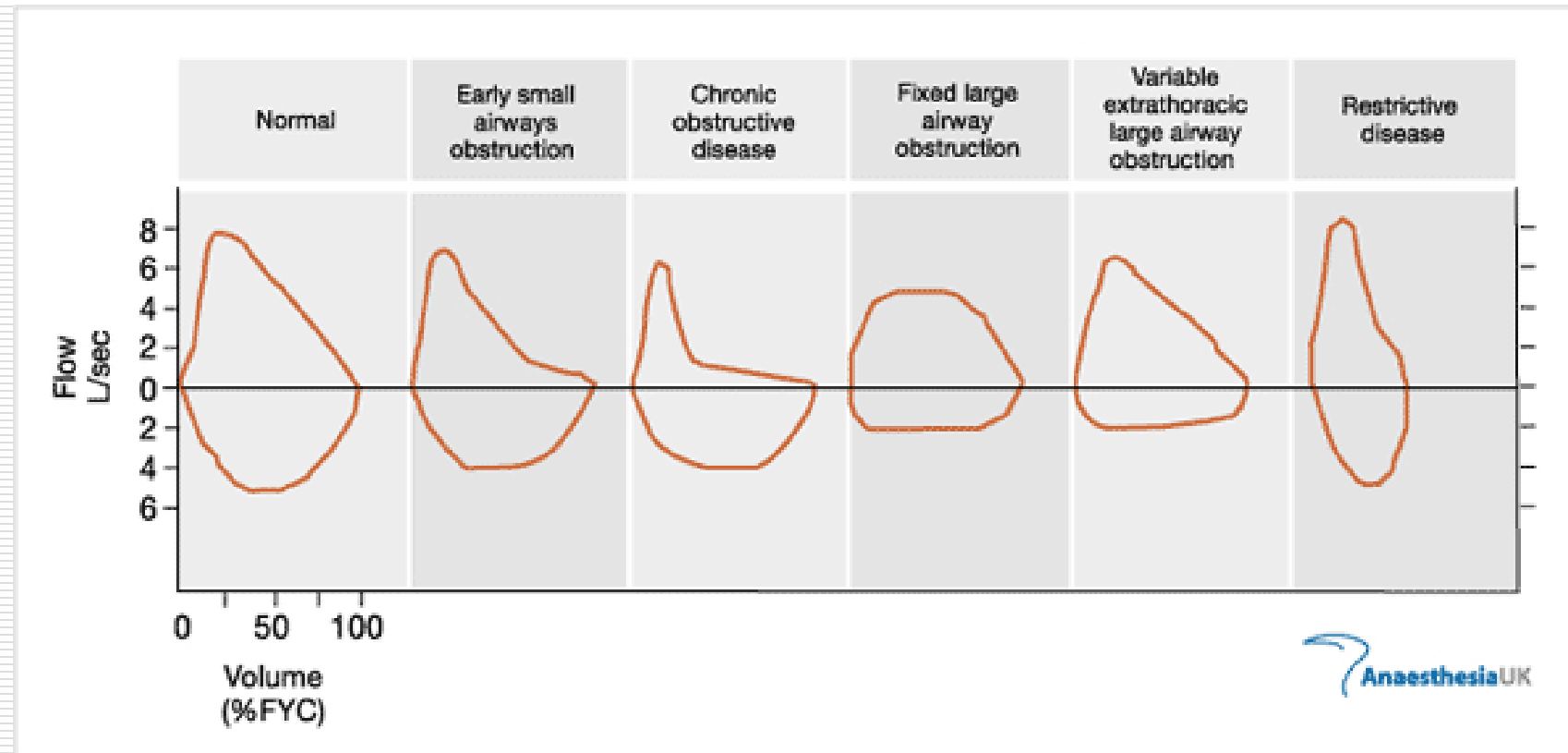
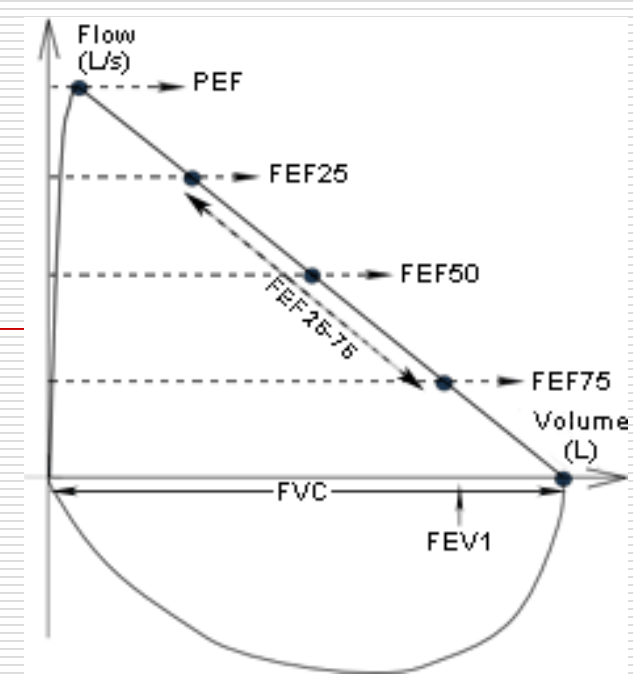


Pathogenesis of Pneumoconiosis

Fibrogenic dust e.g silica and asbestos produce progressive fibrosis in the lung

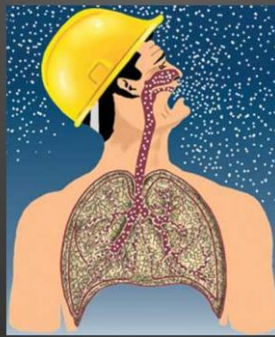


Lung function assessment



PNEUMOCONIOSIS

- ***effects of accumulation of mineral dusts in the lungs;***
- ***the typical reaction is fibrosis***
- Risk and extent
 - intensity and cumulative amount of exposure over time
- Prevention
- occupational safeguards
- case of asbestos, legislative bans on use
 - no effective treatments for these diseases



Silica and Silicosis

<http://safetysolutionsatwork.com/blog/wp-content/uploads/2012/01/silica-lung1.jpg>

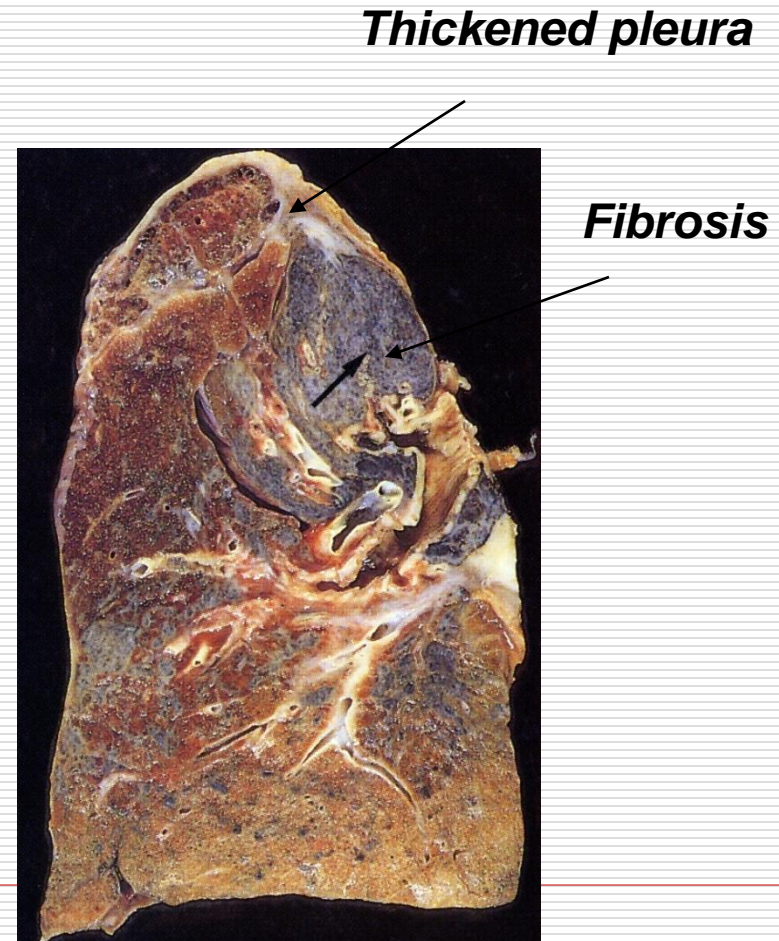
SILICOSIS



- Exposure to crystalline-free silica
- Inflammatory and fibrotic reaction
- Silicotic nodule
- Occupations
 - Mining
 - Stone cutting
 - Carving
 - Polishing
 - Foundry work
 - Abrasive clearing (e.g., sandblasting)
- exposure is usually chronic (over years)
- acute disease in the setting of heavier short-term exposures

Silicosis

- ❑ *Silicosis is **permanent scarring of the lungs** caused by inhaling silica (quartz, SiO_2) dust.*
- ❑ *It is a slowly progressive, nodular, fibrosing pneumoconiosis*



Occupational Exposure to Silica

- *Silicosis, the oldest known occupational lung disease, develops in people who have inhaled silica dust for many years.*
- *Silica is the main constituent of sand, so exposure is common among:*
 - *metal miners,*
 - *sandstone and granite cutters,*
 - *foundry workers,*
 - *and potters.*



Clinical Varieties of silicosis

☐ **Chronic silicosis**

- *Commonest form*
- *Nodular opacities*
- *Appears after 15 years or more of exposure*

☐ **Accelerated silicosis**

- *Appears after 5 – 10 years*
- *Similar to Chronic form*
- *Due to exposure to high concentration of silica*

☐ **Acute silicosis**

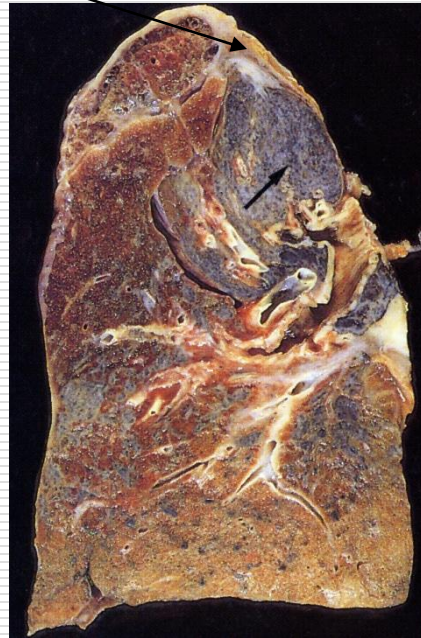
- *Due to massive exposure to very fine silica particles*
- *It causes alveolar proteinosis , fatal, appears within 5 years of exposure*

Pathogenesis of silicosis

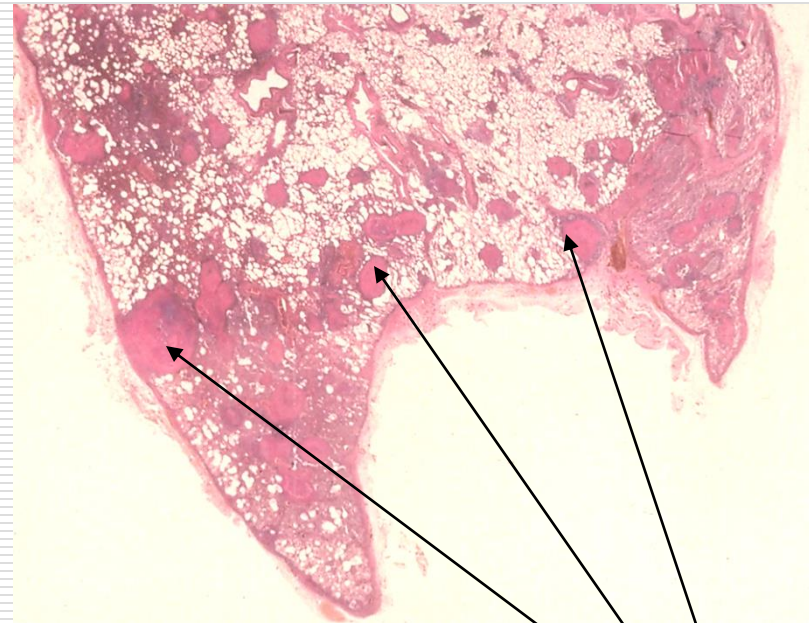
- ☐ *When inhaled, silica dust passes into the lungs, and scavenger cells such as macrophages engulf it.*
 - ☐ *Enzymes released by the scavenger cells cause the lung tissue to scar.*
 - ☐ *At first, the scarred areas are **tiny round lumps** (simple nodular silicosis), but eventually they may combine into larger masses (complicated silicosis, PMF).*
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Pathology of Silicosis

Thickened pleura



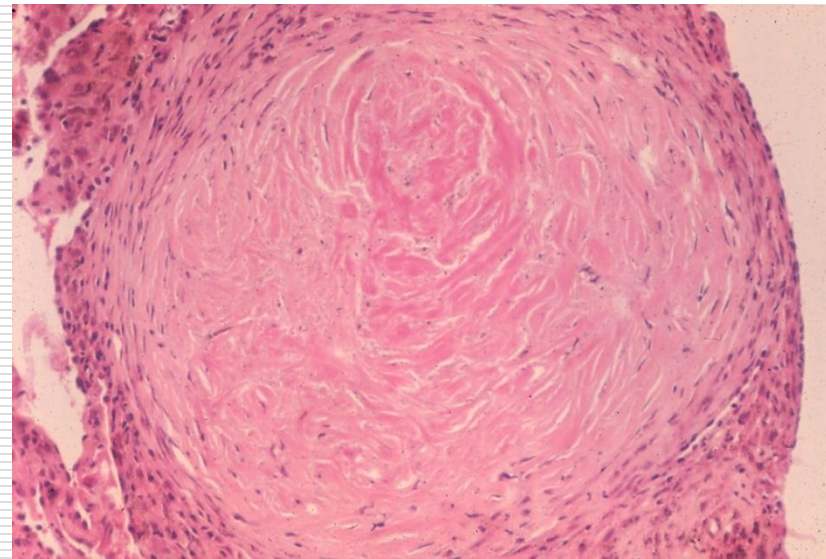
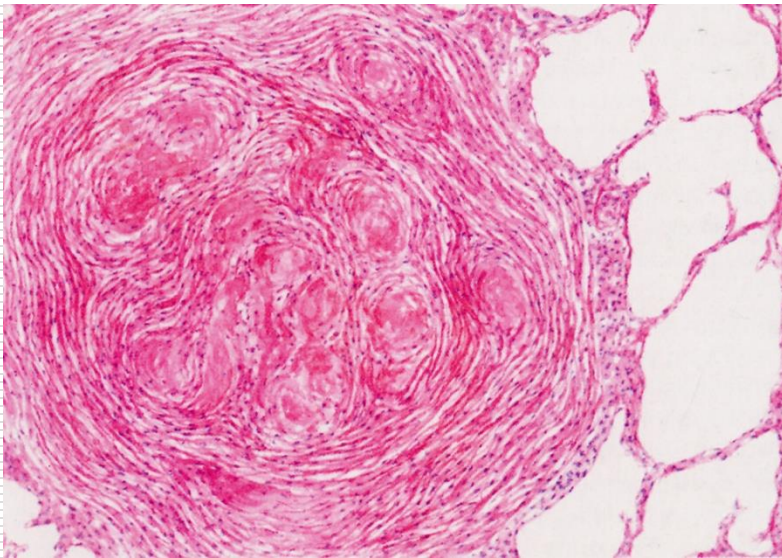
Lung fibrosis



Silicotic nodules

Pathology of silicosis

Silicotic nodule



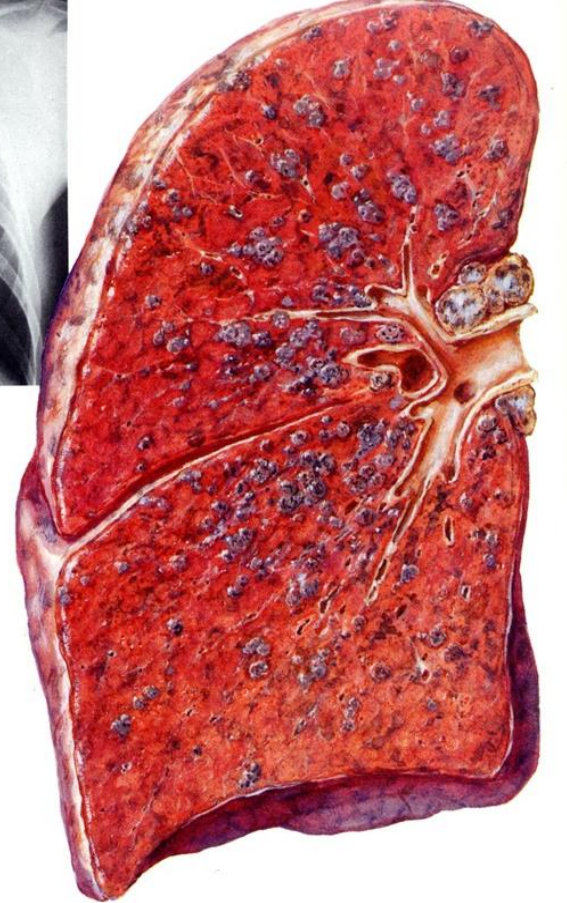
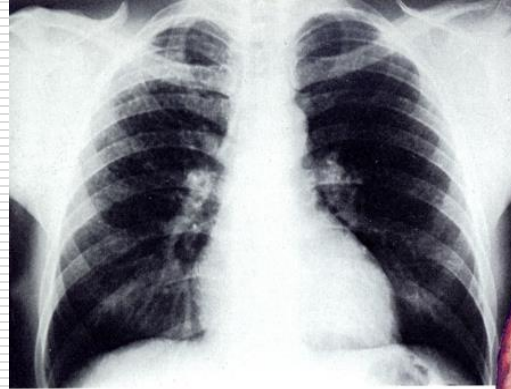
(Onion shape appearance)

*Early lesions are nodules
More fibrous tissue, less cells*

*It is composed of lamellae of collagen
It contains dust particles, carbon*

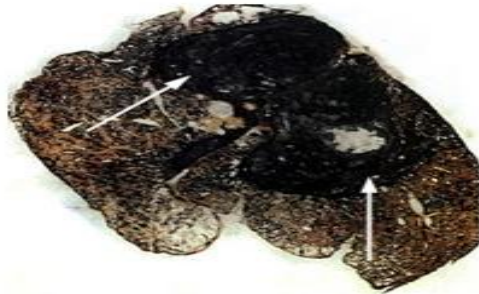
Pathology of silicosis

Initial lesions start first in the middle and upper parts of the lung as small tiny nodules, then spread to the rest of the lung



Symptoms and Diagnosis

Lung Specimen: Silicosis



Lung Specimen: Normal



An abnormal cross- section specimen of the lung shows large black nodules (arrows). This finding is seen in silicosis caused by exposure to silica dust. A normal cross-section specimen of the lung is shown on the right for comparison

- *Usually, symptoms appear only **after 20 or more years of exposure to the dust.***
- *However, in occupations such as sandblasting, tunneling, and manufacturing abrasive soaps, in **which high levels of silica dust are produced, symptoms may appear in less than 10 years***

Symptoms and Diagnosis

- *The least serious type of lung disease from silica is **simple nodular silicosis**.*
 - *People with simple nodular silicosis usually have no trouble breathing, **but they may cough (dry)**.*
 - *They may **produce sputum** (also called phlegm) when their large airways are **inflamed due to infection or due to smoking**.*
-

Symptoms and Diagnosis

- *People who have the more serious type, **complicated silicosis**, may cough, produce sputum, and have severe shortness of breath.*
 - *At first, the shortness of breath may occur only during exercise, but eventually it occurs even during rest.*
 - *Breathing may worsen for years after the person stops working with silica.*
-

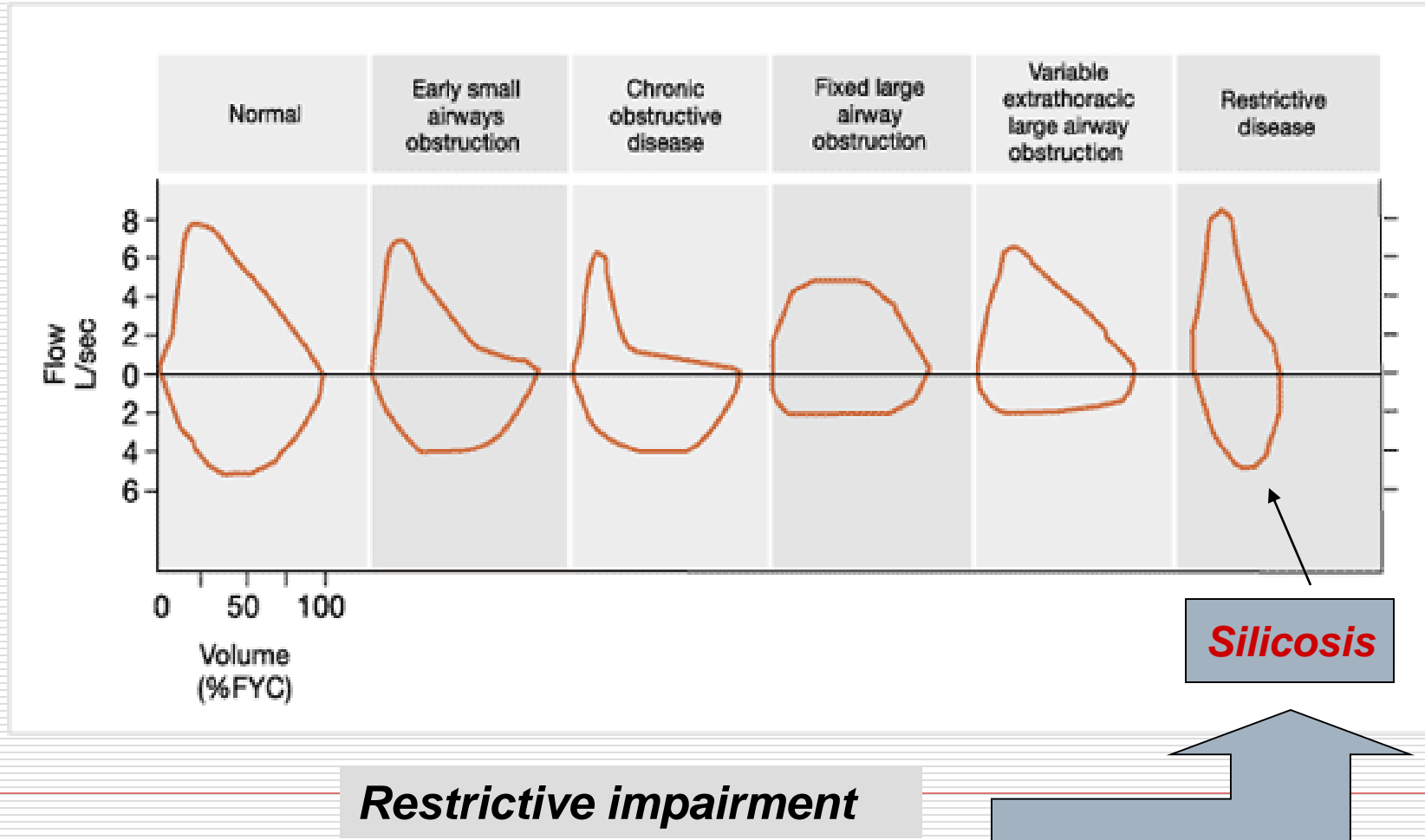
Symptoms and Diagnosis

- *The lung damage strains the right side of the heart and can lead to a type of **heart failure** (called **cor pulmonale**), a condition which may be fatal*
 - *Also, when exposed to the organism that causes tuberculosis, **people with silicosis are many times more likely to develop tuberculosis than people without silicosis.***
-

Diagnosis of Silicosis

- ☐ *Positive History of exposure to free silica dust*
 - ☐ *Early dry irritant cough*
 - ☐ *Later, shortness of breath*
-

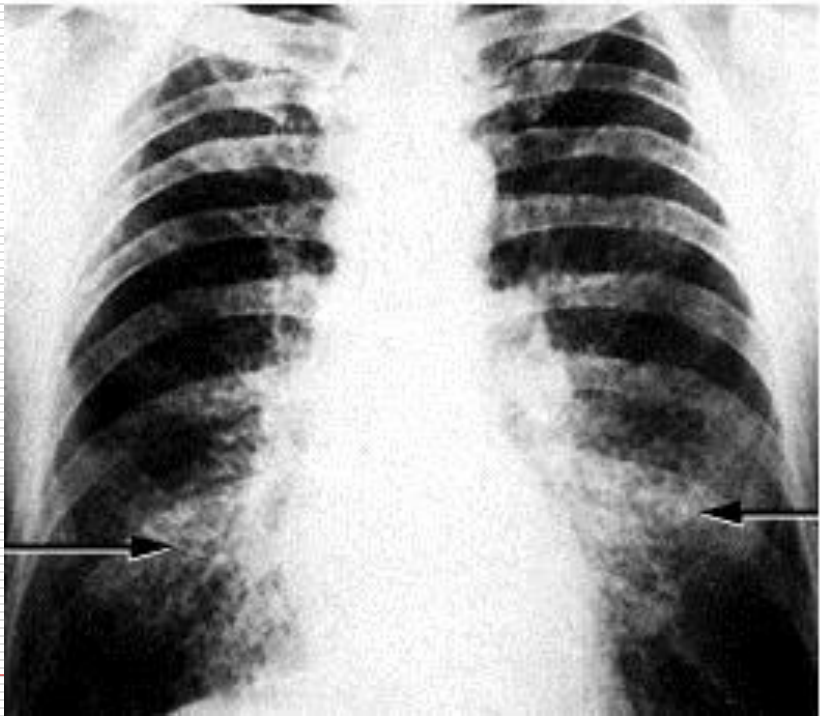
Diagnosis of Silicosis: Lung function



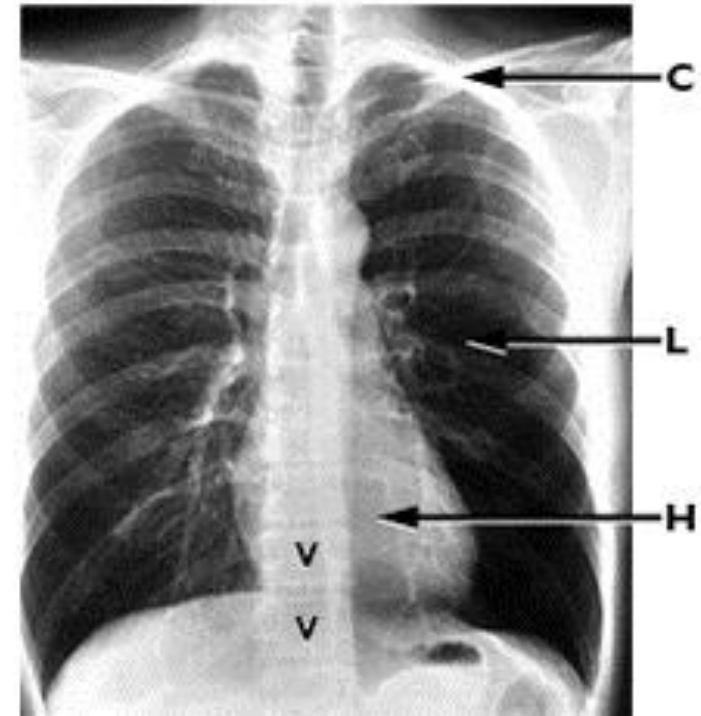
Diagnosis of Silicosis: CXR

□ Nodular opacities

Chest X-Ray: Silicosis

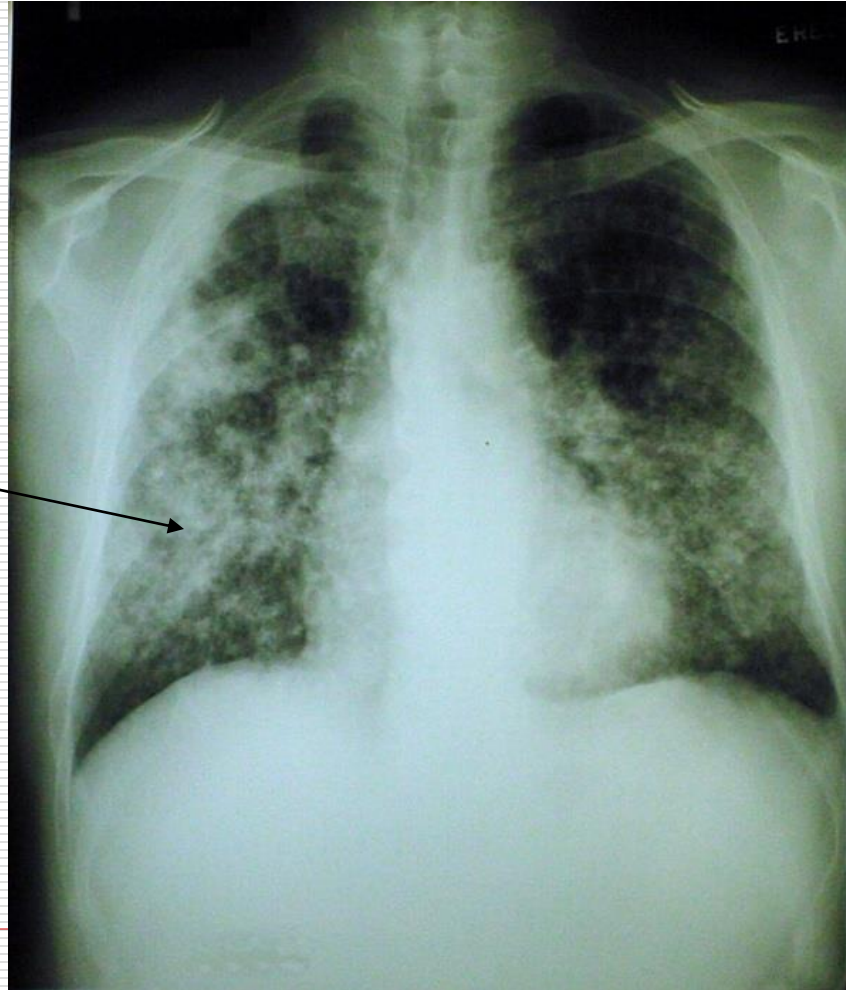


Normal X-Ray



Diagnosis of Silicosis: CXR

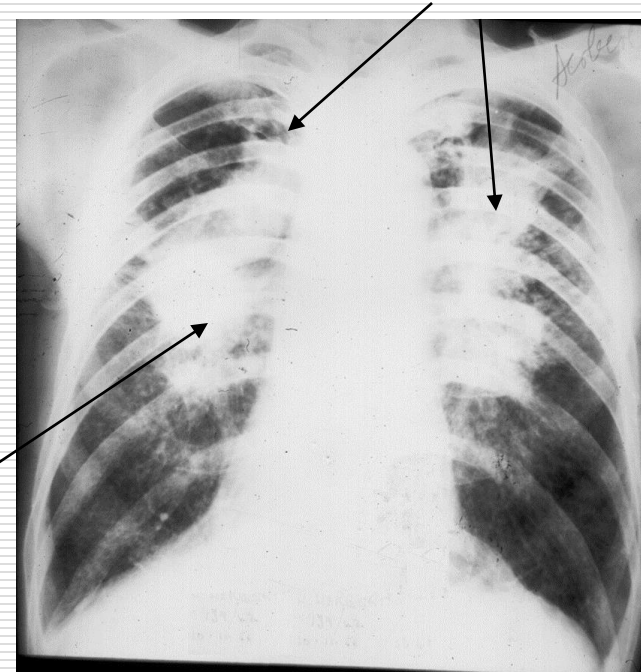
Nodular opacities



Diagnosis of Silicosis: CXR



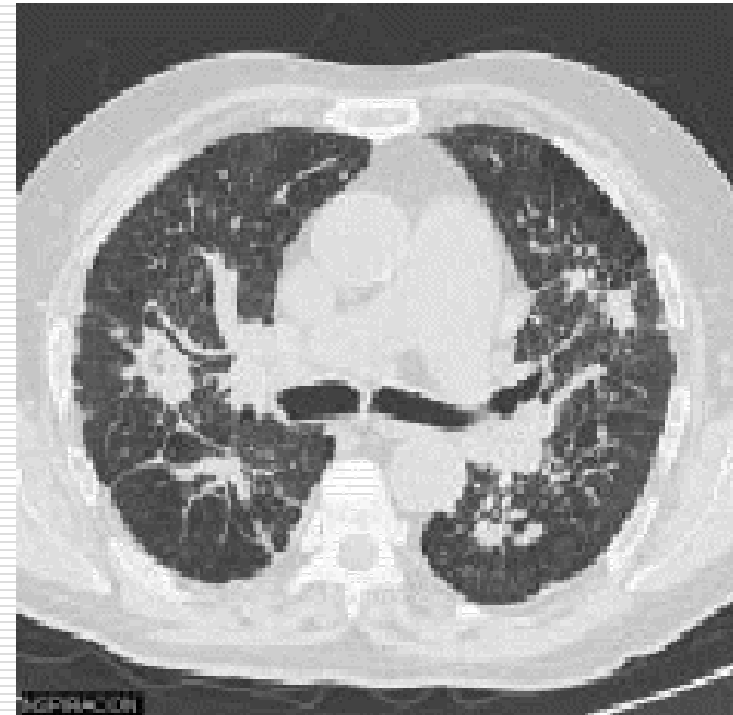
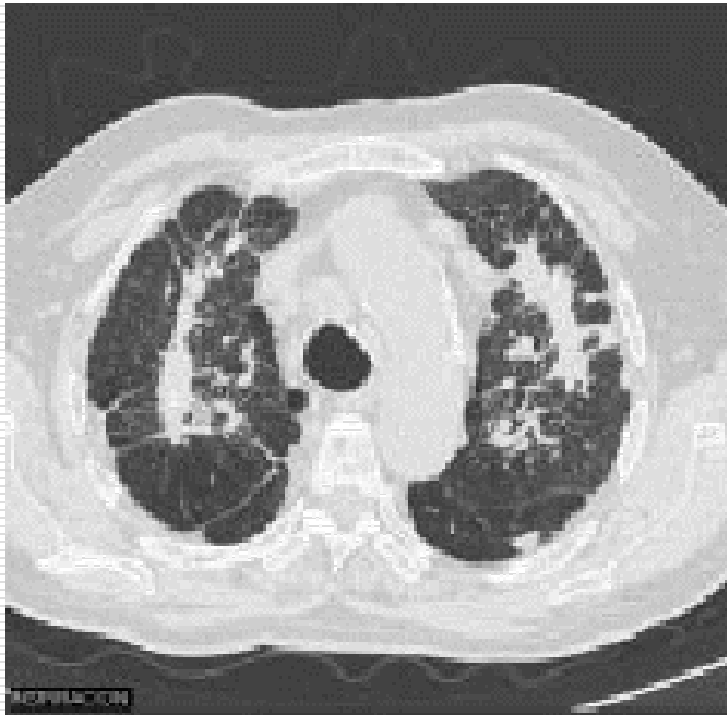
PMF



Angle's wings

Coalescence of opacities

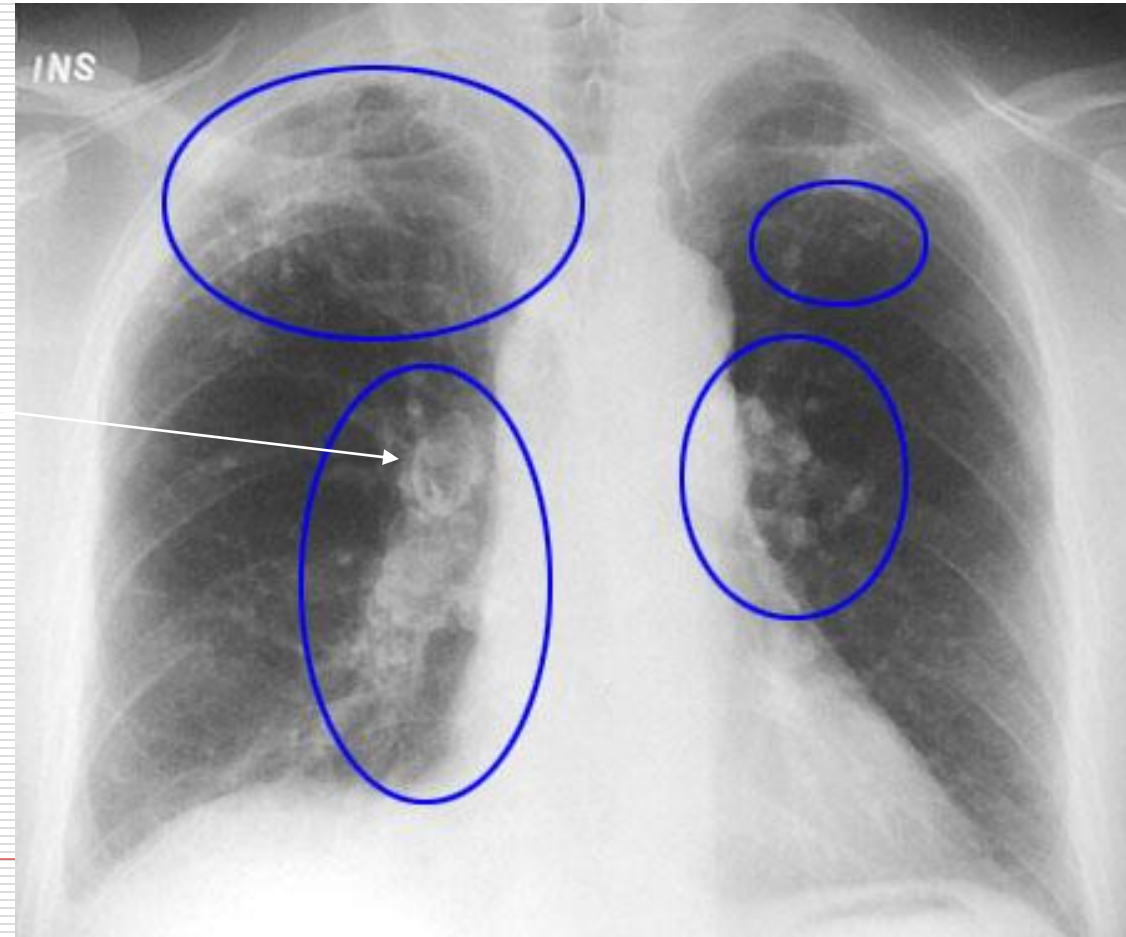
Diagnosis of Silicosis: CXR



HRCT

Diagnosis of Silicosis: CXR

***Egg shell appearance
of hilar lymph nodes***



Diagnosis of Silicosis: CXR

- *Tuberculosis is the most common complications among workers with silicosis*



Prevention and Control

- *Controlling silica dust in the workplace is **key to preventing silicosis.***
 - *When dust cannot be controlled, as may be true in the sandblasting industry, **workers should wear protective gear,** such as hoods that supply clean external air or special masks that efficiently filter out the tiny particles.*
 - *Such protection may not be available to all people working in a dusty area (for example, painters and welders), **so whenever possible, abrasives other than sand should be used.***
-

Prevention and Control

- *Workers exposed to silica dust should have **regular chest x-rays** —every 6 months for sandblasters and every 2 to 5 years for other workers—so that problems can be detected early.*
 - *If the x-rays show silicosis, a doctor will probably advise the worker to **avoid continued exposure to silica***
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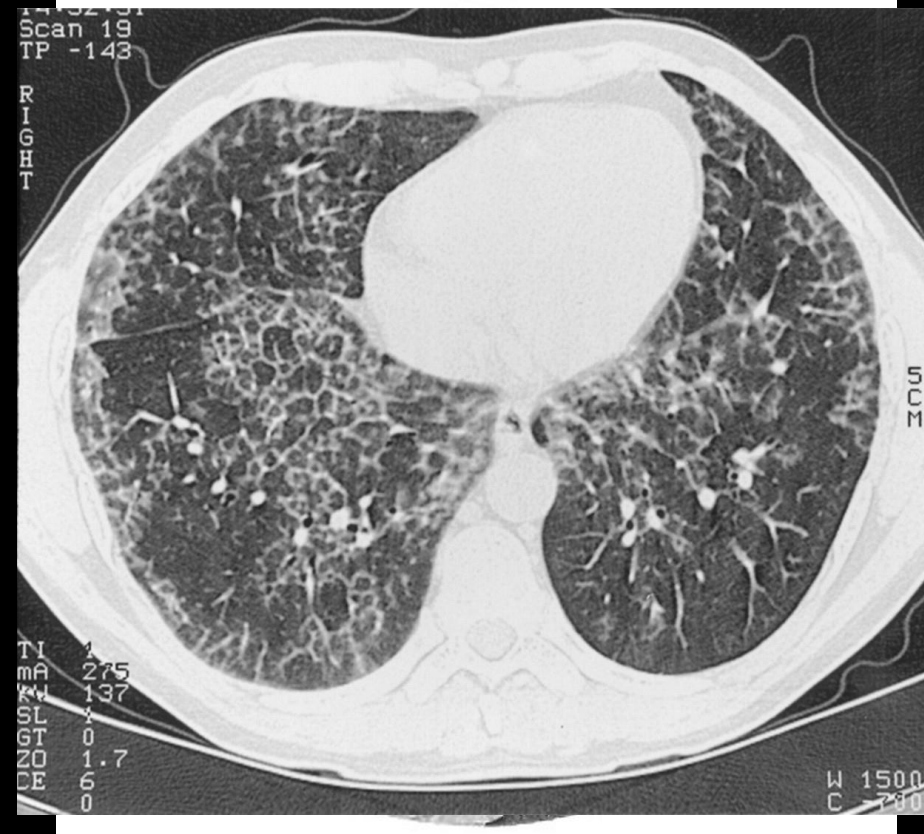
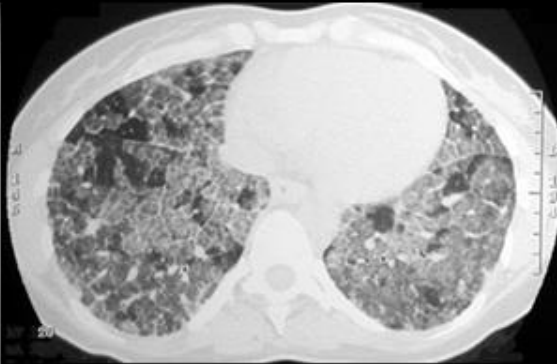
Treatment

- *Silicosis cannot be cured, but its progression can be slowed if exposure to silica is avoided, especially at an early stage of the disease.*
 - *A person who has difficulty breathing may benefit from the treatments used for chronic obstructive pulmonary disease, such as drug therapy to keep the airways open and free of mucus.*
 - *Because people with silicosis have a high risk of developing tuberculosis, they should have regular checkups that include a tuberculosis skin test*
-

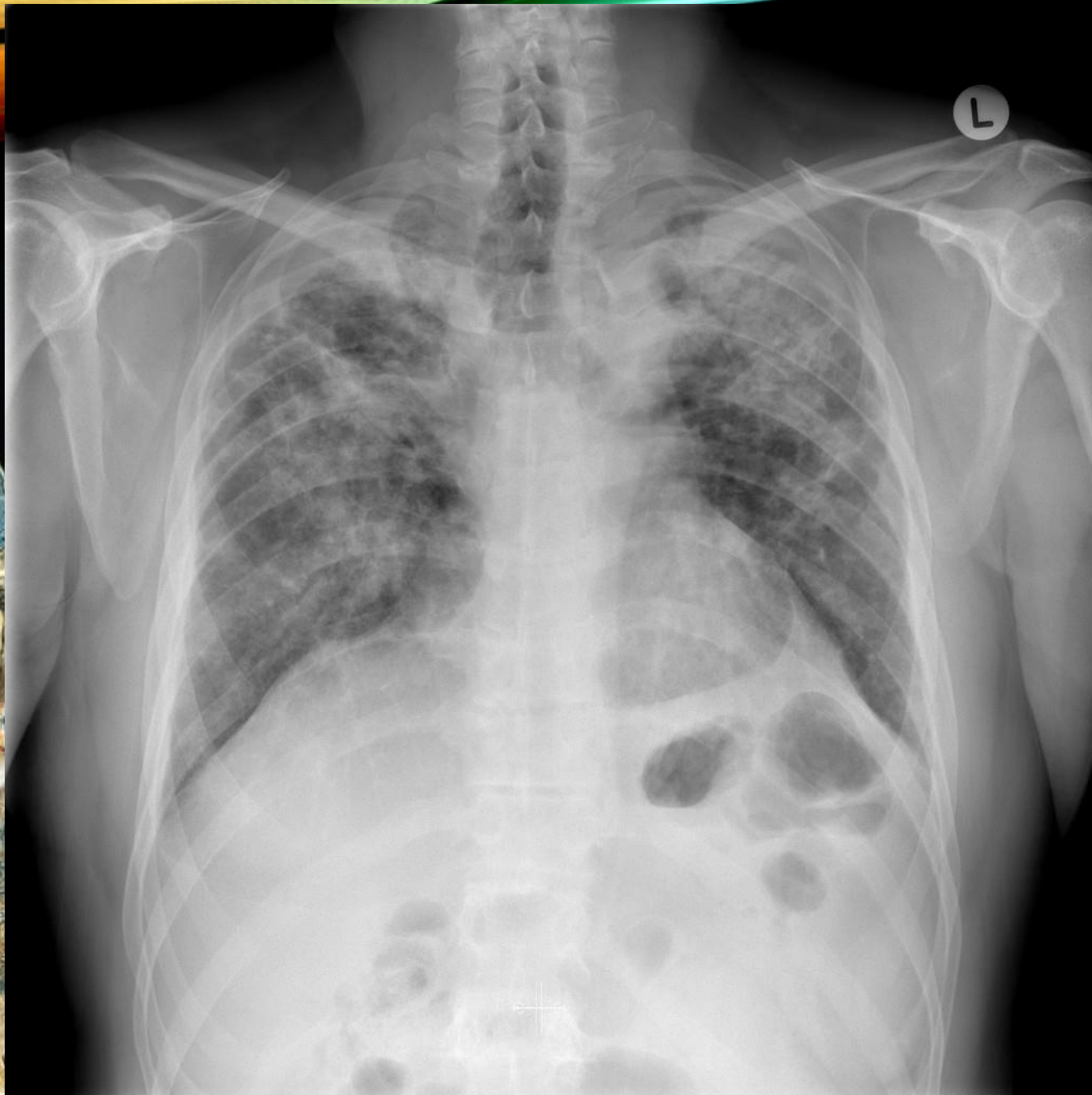
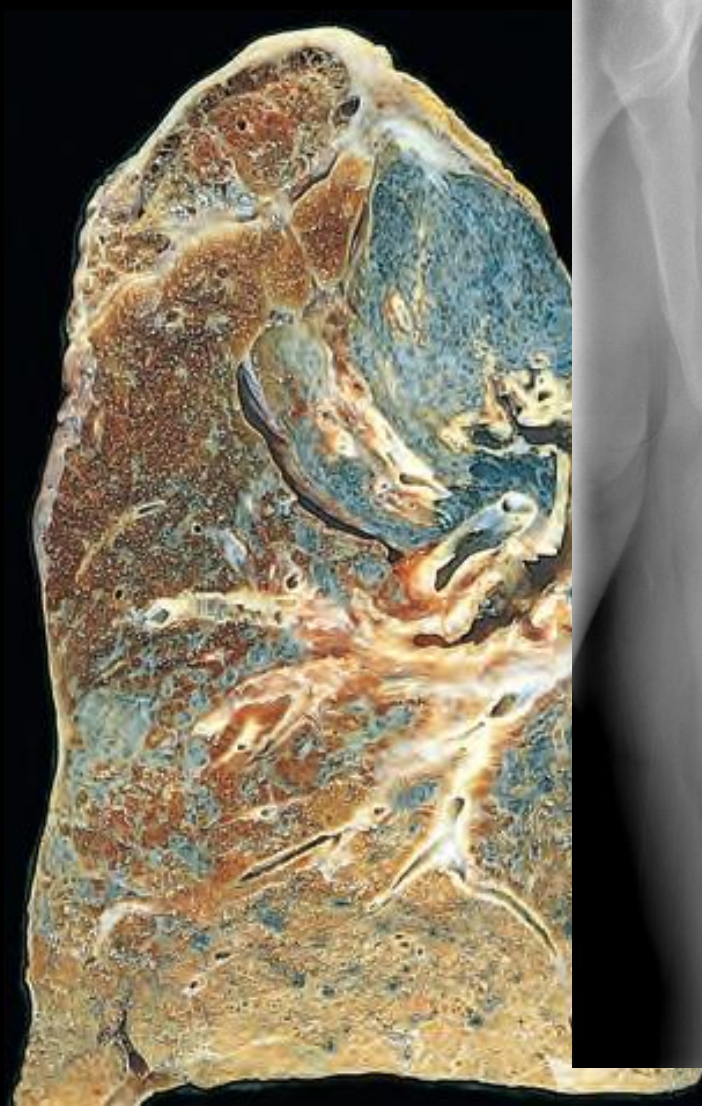


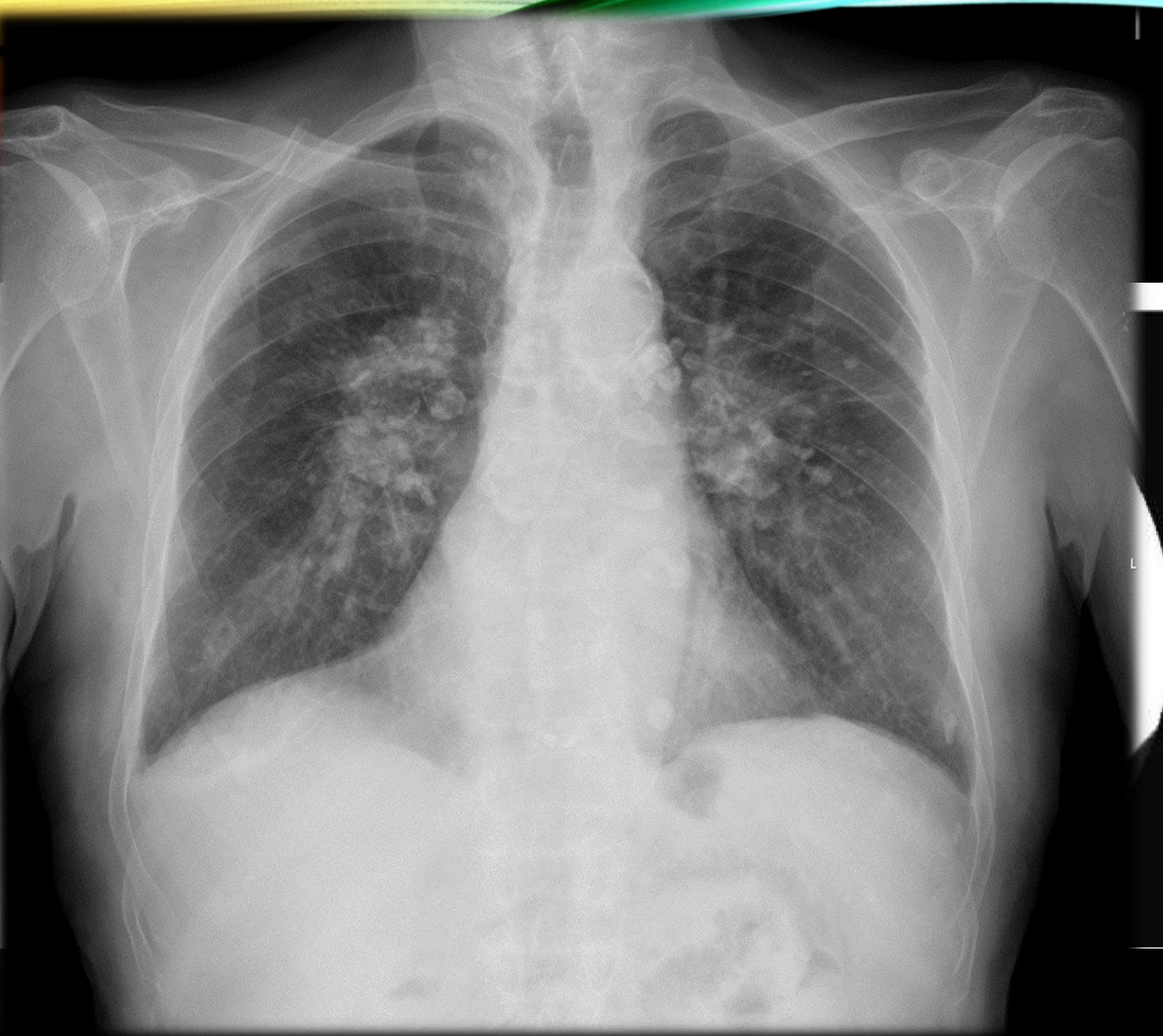
ACUTE SILICOSIS

- pulmonary alveolar proteinosis
 - accumulation of surfactant in the alveolar spaces



little as 10 months of exposure





SILICOSIS

PULMONARY FUNCTION TESTS

- in simple nodular silicosis
 - normal
 - mixed obstructive or restrictive pattern
- progressive massive fibrosis
 - severe restriction and hypoxemia
- **elevated risk for tuberculosis**
 - **screened for latent tuberculosis infection**
- *association between silicosis and rheumatoid arthritis*
- *and scleroderma*

COAL WORKER'S PNEUMOCONIOSIS

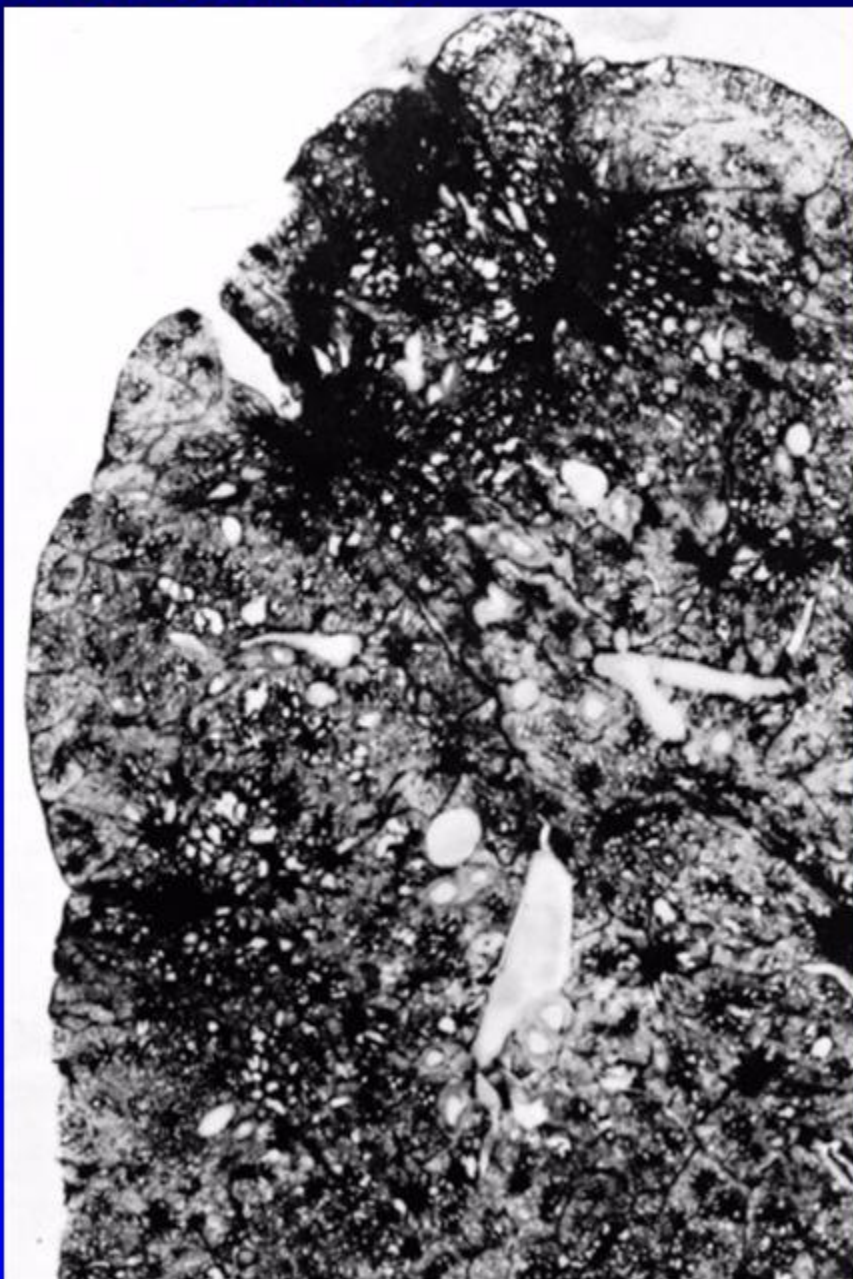
- uncommon cause of pulmonary fibrosis
- **coal dust and graphite**
- underground mines
- formation of pigmented lesions in the lung surrounded by emphysema,
 - coal macules
- Progressive massive fibrosis
- chronic cough usually productive
 - bronchitis related to coal exposure or to tobacco
- chest radiograph shows
 - diffuse, small, rounded opacities
- with silicosis, there is an association with rheumatoid arthritis.

Coal worker pneumoconiosis

Morphology:

•**Complicated CWP:**

- Black scars exceed 2 cm in diameter some times up to 10 cm
- It consists of dense collagen and carbon pigments.
- Cor pulmonale.
- Miners who have rheumatoid arthritis and PMF are called Caplan's syndrome.



CAPLAN'S SYNDROME

- multiple, large, sometimes cavitary lung nodules
- Seropositive rheumatoid arthritis

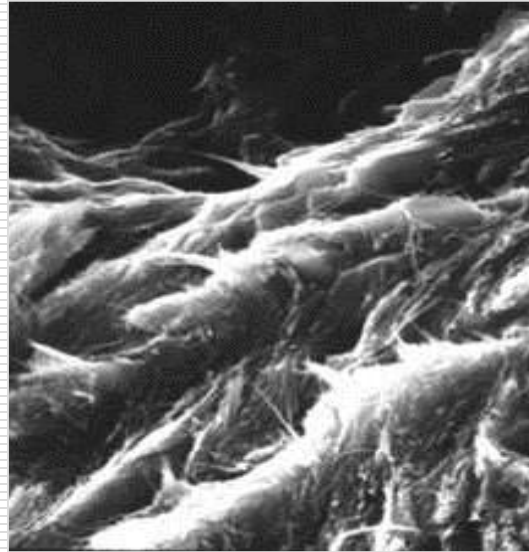


Caplan's
syndrome

- **after coal dust exposure**

first described in coal miners
But subsequently in patients with silicosis

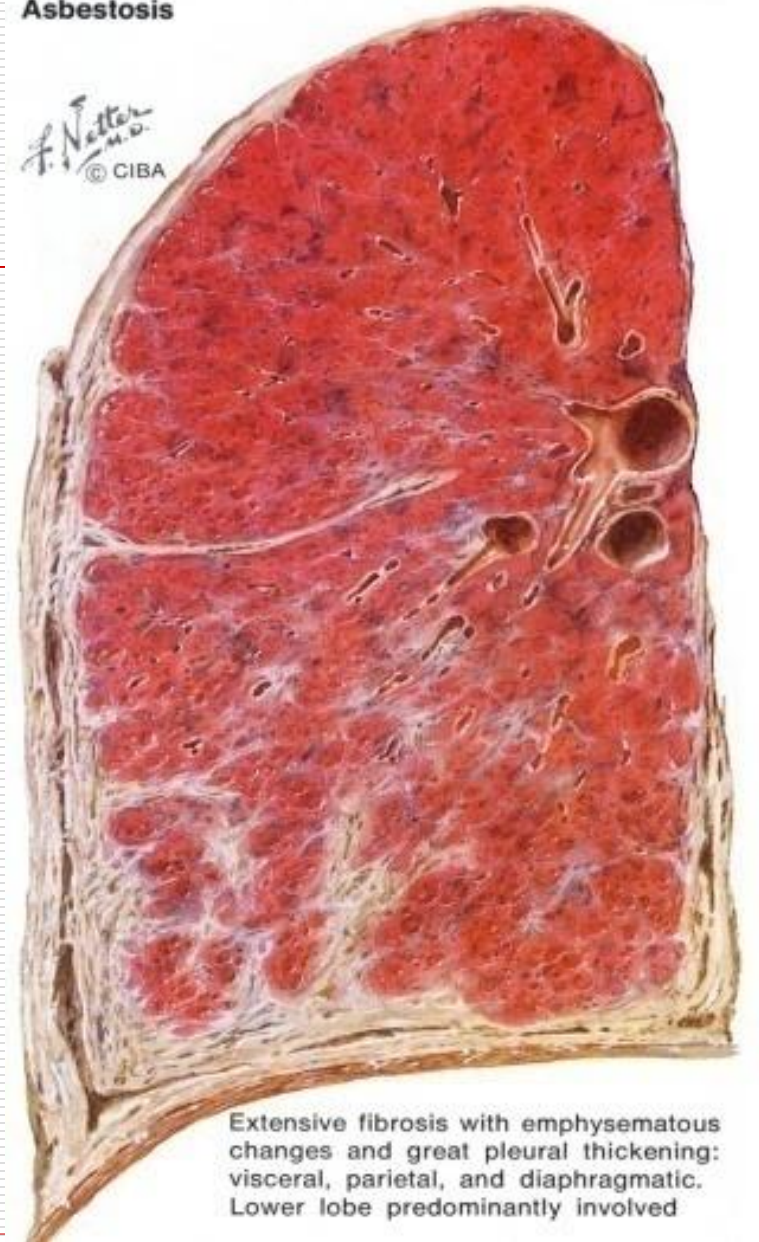
Asbestosis



Asbestos fibers

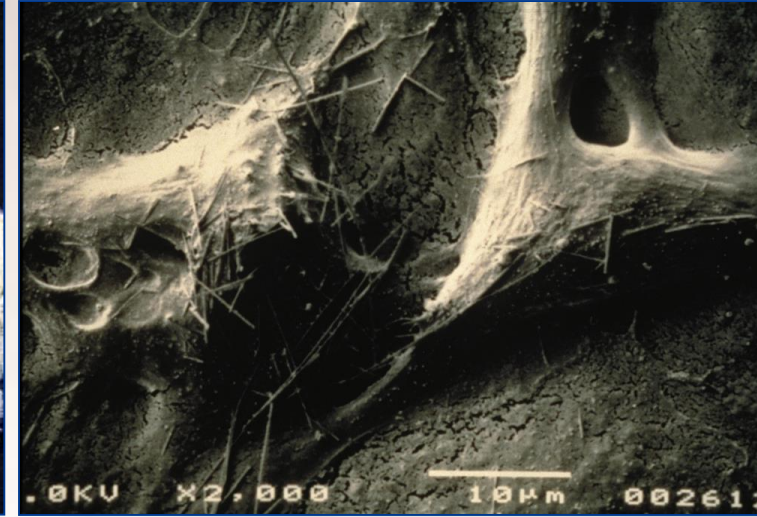
Asbestosis

F. Netter M.D.
© CIBA



Extensive fibrosis with emphysematous changes and great pleural thickening: visceral, parietal, and diaphragmatic. Lower lobe predominantly involved

Asbestos: Types



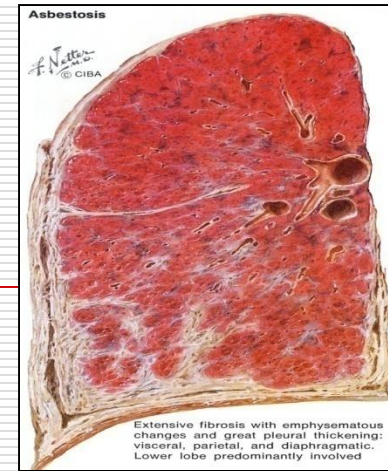
Serpentine (93% of commercial use)	Amphibole (7% of commercial use)
Chrysotile	Actinolite, Amosite, Anthophyllite, Crocidolite, Richterite, Tremolite

Asbestos



Asbestosis

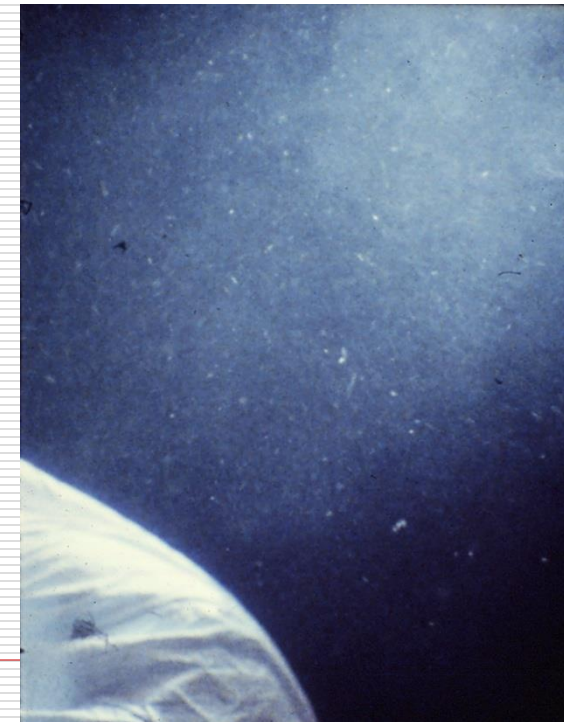
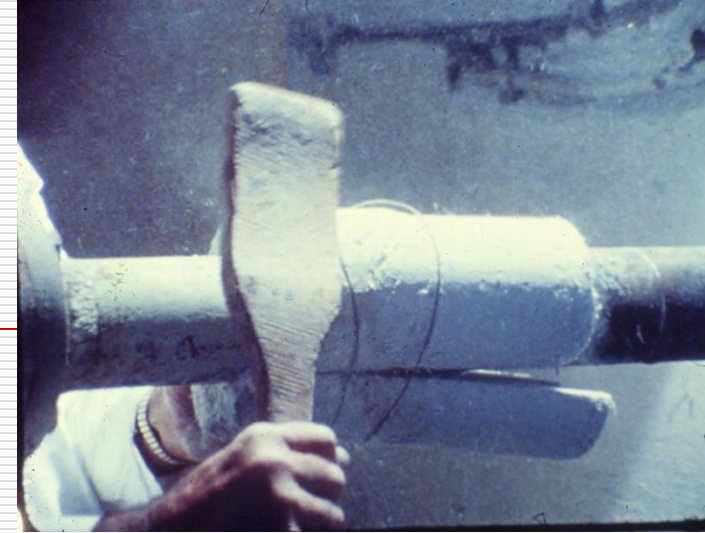
- ☐ *It is diffuse fibrosis of the lung parenchyma*
- ☐ *Asbestos fibers are highly resistant to heat, acids and chemicals*
- ☐ *Used to be widely used in industries*
- ☐ *Banned from use globally since 1973 because it is high carcinogenic*



Occupational exposure

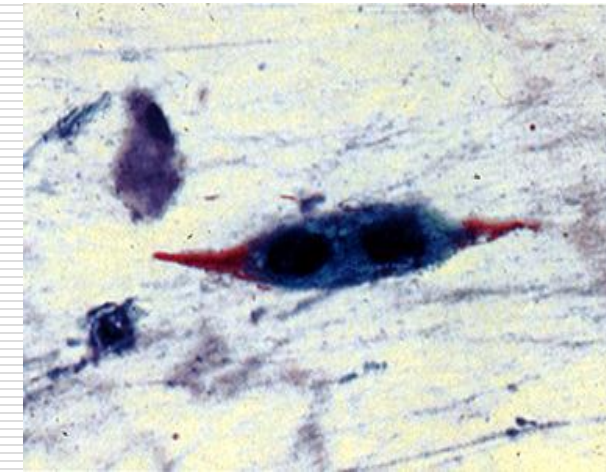
- ☐ *Workers in car industry (brake manufacture)*
- ☐ *Workers making insulation*
- ☐ *Workers in ship building*
- ☐ *Workers who **demolish buildings** that have **insulation containing asbestos** are at increased risk.*

- ☐ *The **more a person is exposed** to asbestos fibers, the greater the risk of developing an **asbestos-related disease**.*



ASBESTOSIS

- *Asbestosis is **widespread scarring of lung tissue** caused by breathing asbestos dust.*
- ***Asbestos** is composed of **fibrous mineral silicates** of different chemical compositions.*
- *When inhaled, asbestos fibers settle deep in the lungs, **causing diffuse fibrosis**.*



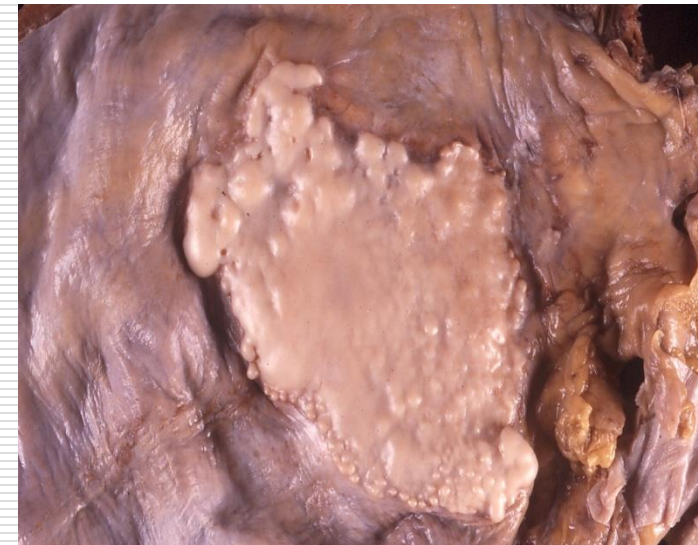
A microscopic specimen of a cell from the lungs shows the presence of an asbestos fiber with a mineral coating. This patient worked in a shipyard for many years

ASBESTOSIS

- ❑ *Asbestos inhalation also can cause the two layers of membrane covering the lungs (the pleura) to thicken; these thickenings are called **pleural plaques**.*
- ❑ *These plaques do not become cancerous*



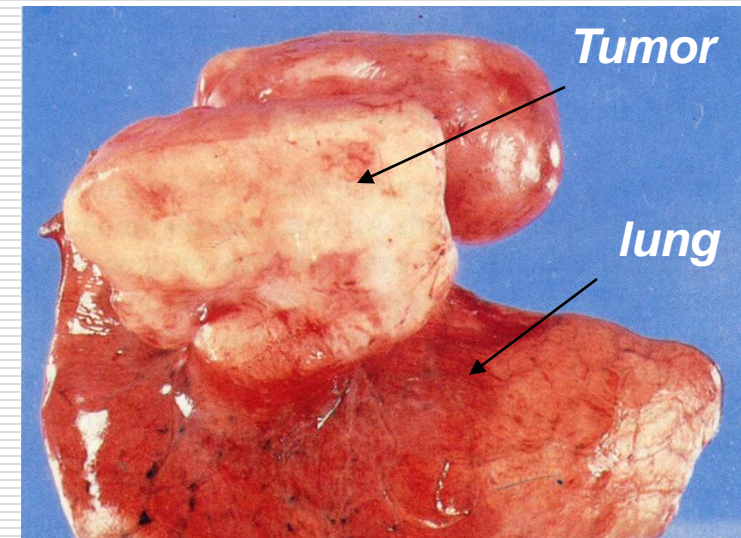
*Macrophage engulfing
asbestos fiber*



Diaphragmatic plaque

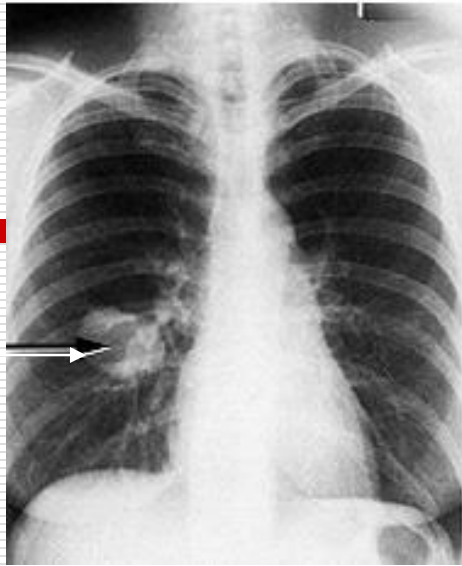
ASBESTOSIS

- ❑ *Asbestos also causes cancer in the pleura, called **mesothelioma**, or in the membranes of the abdomen, called **peritoneal mesothelioma**.*
- ❑ *In the United States, asbestos is the only known cause of cancerous (malignant) mesothelioma.*
- ❑ *Smoking is not a cause of cancerous mesothelioma*



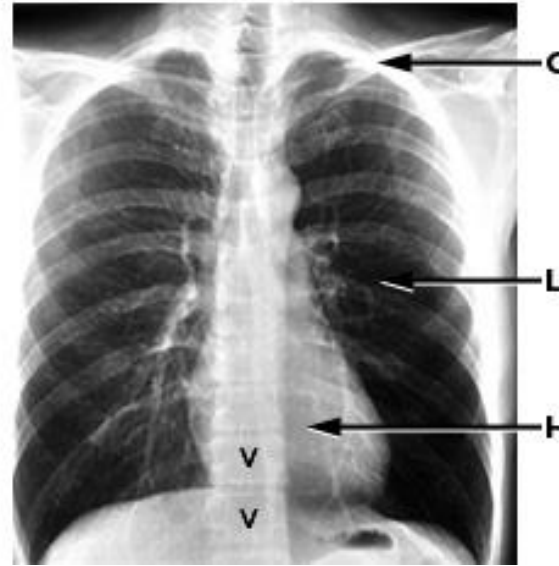
Mesothelioma

Lung Tumor



cancer

Normal X-Ray



- *An abnormal chest x-ray shows a tumor (arrow) in the lung.*
- *A normal chest x-ray is shown on the right for comparison; the heart (H), lungs (L), vertebrae (v), and collarbone (C) can be seen.*

- *Asbestos can also cause **lung cancer**. Lung cancer from asbestos is related in part **to the level of exposure to asbestos fibers**.*
- *however, among people **with asbestosis, lung cancer occurs most commonly in those who also smoke cigarettes**, particularly those who smoke more than a pack a day .*

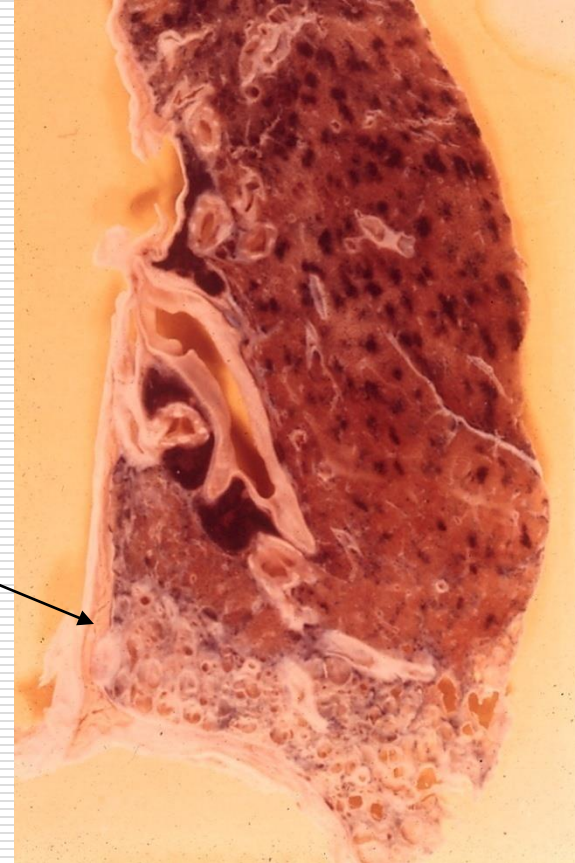
Pathology of Asbestos Related Diseases

- Diffuse interstitial fibrosis starting at the base of the lung
- Thickening of the pleura



Pathology of Asbestos Related Diseases

**Diffuse fibrosis
starting at the base of the lung
“Honey comb appearance”**

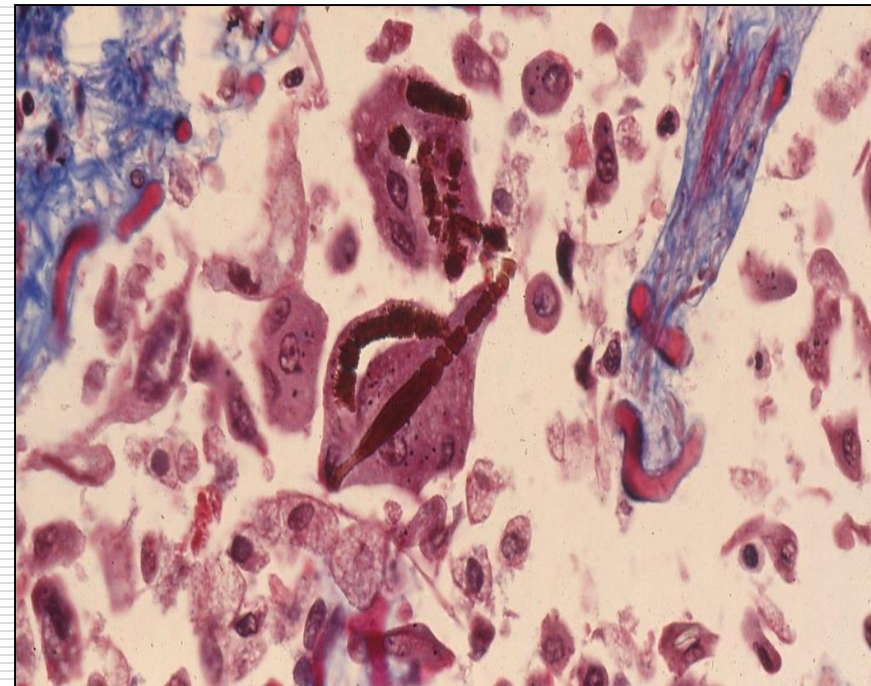


Pathology of Asbestos Related Diseases

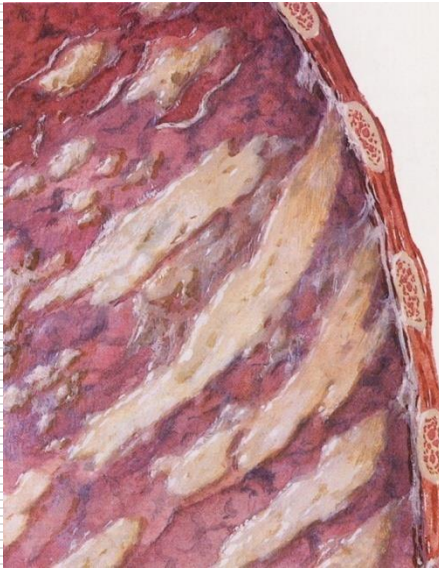
Asbestos bodies:

Fibers may be coated with hemosiderin and undergoes degeneration and form bodies

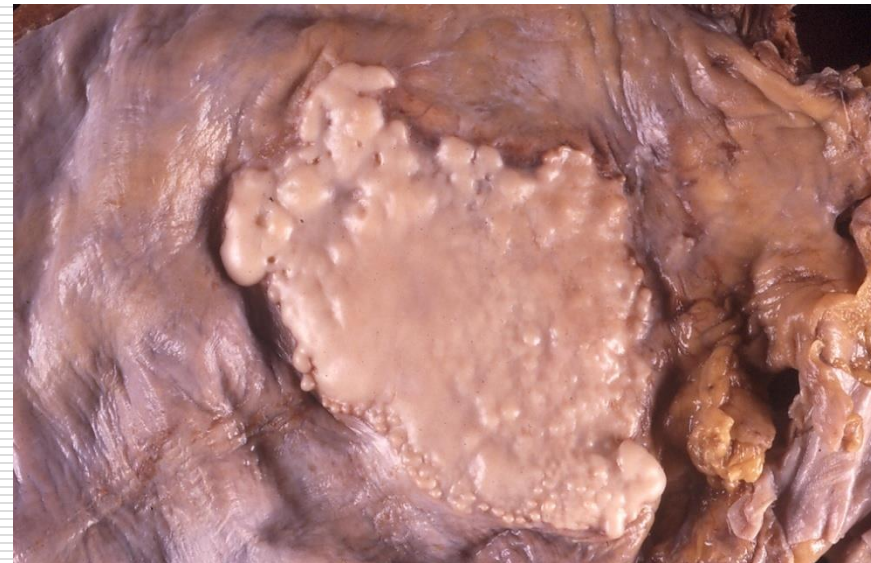
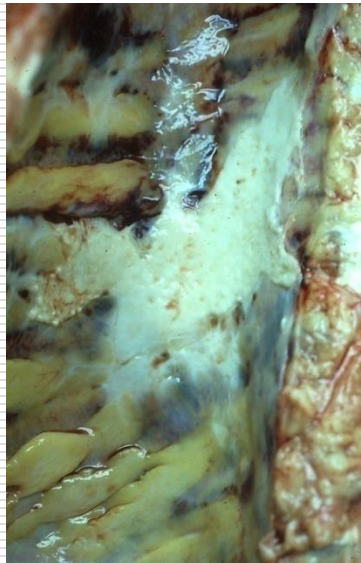
- *Drum stick appearance*
- *Rods*
- *Dumple shaped*



Pathology of Asbestos Related Diseases



Parietal pleural plaques



Diaphragmatic pleural plaques

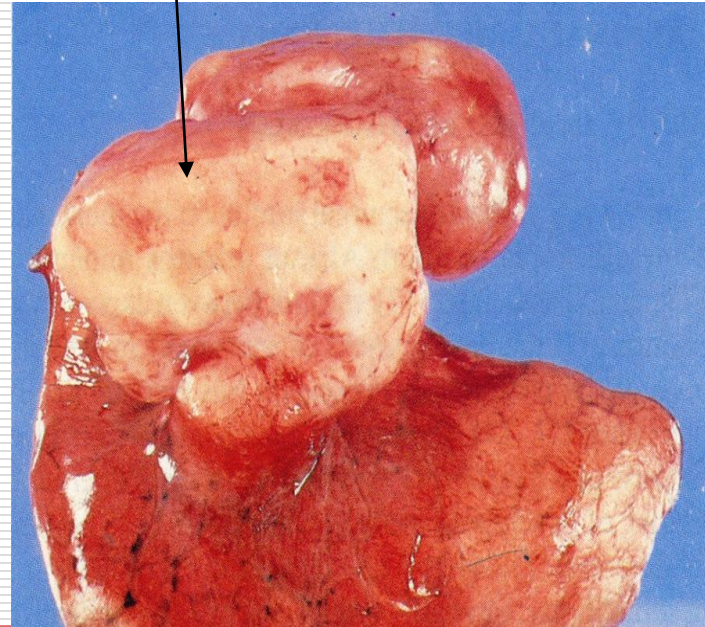
Usually asymptomatic

Pathology of Asbestos Related Diseases

**Bronchogenic
Carcinoma**



Mesthothelioma



Pathology of Asbestos Related Diseases

**Clubbing of the Fingers
which reflects chronic
hypoxic lung disease**



Asbestos related diseases

□ *Diseases are:*

- *Interstitial pulmonary fibrosis (asbestosis)*
 - *Bronchogenic carcinoma*
 - *Pleural Effusions*
 - *Fibrous plaques*
 - *Pleural fibrosis*
 - *Mesotheliomas (malignant tumor of pleura and peritoneum)*
 - *Laryngeal (colon?) neoplasms*
-

Asbestos-Associated Diseases

- ***Respiratory diseases:***
 - *Parenchymal asbestosis*
 - *Asbestos-related pleural abnormalities*
 - *Lung carcinoma*
 - *Pleural mesothelioma*
- ***Nonrespiratory diseases:***
 - *Peritoneal mesothelioma*
 - *Possibly, other extrathoracic cancers*
 - *Rarely, cor pulmonale or constrictive pericarditis*

Symptoms

- ❑ *Symptoms of asbestosis appear **gradually** only **after large areas of the lung become scarred**.*
 - ❑ *The scarring causes the lungs to **lose their elasticity**.*
 - ❑ *The first symptoms are a **mild shortness of breath** and a **decreased ability to exercise**.*
-

Diagnosis of asbestosis

- ☐ *Positive occupational history of exposure to asbestos dust*
- ☐ *Shortness of breath*
- ☐ *Cyanosis*
- ☐ *Clupping of the fingers*
- ☐ *End inspiratory crepitation*



Parenchymal Asbestosis

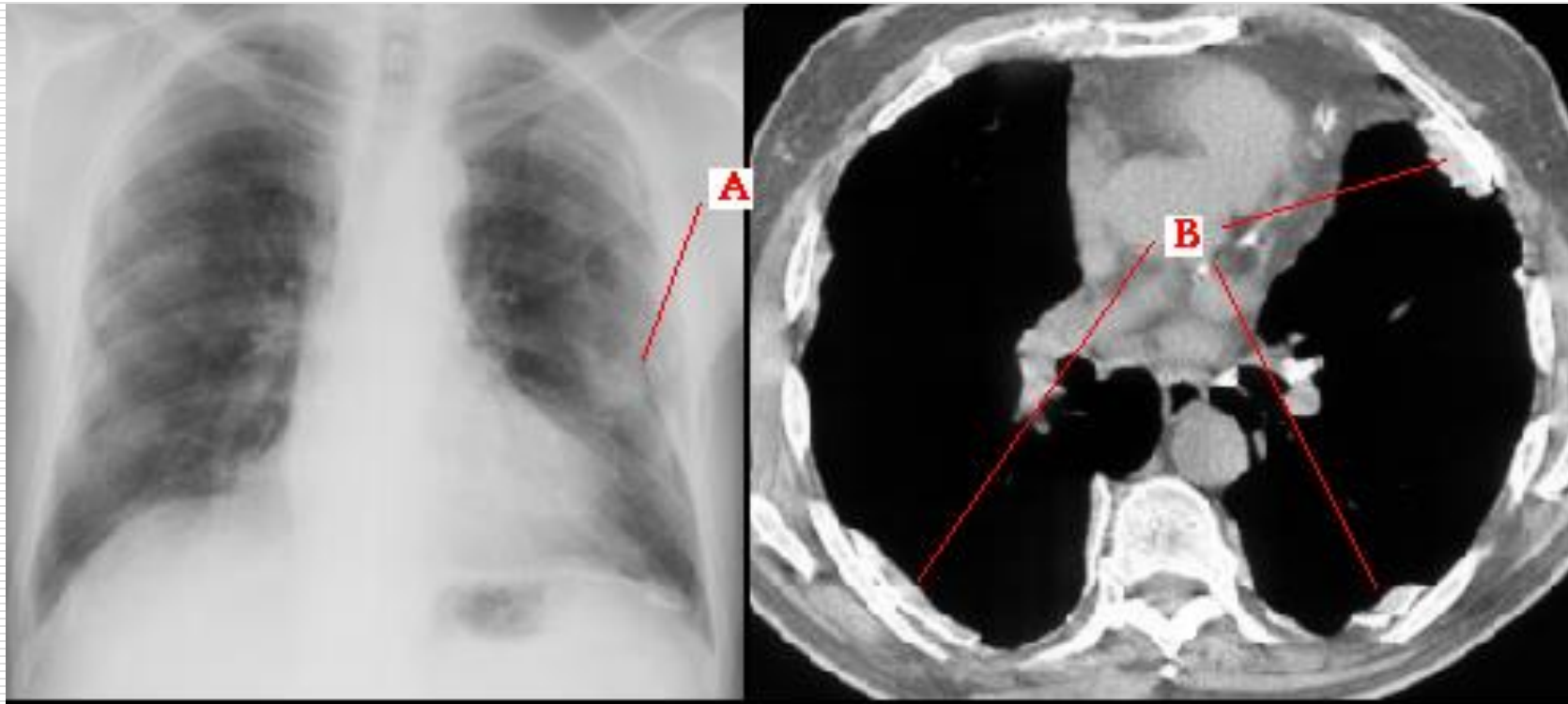
- ***Diffuse interstitial fibrosis with:***
 - ***Restrictive pattern of disease on pulmonary function testing (but can see mixed pattern)***
 - ***Impaired gas exchange***
 - ***Progressive exertional dyspnea***
- ***Radiographic changes: >10 years***
- ***Latency period: 20-40 years***

Asbestos-Related Pleural Abnormalities

- ***Four types of abnormalities:***
 - ***Pleural plaques***
 - ***Benign asbestos pleural effusions***
 - ***Diffuse pleural thickening***
 - ***Rounded atelectasis***
- ***Mostly asymptomatic, though some can cause dyspnea or cough***
- ***Latency periods: 10-30 years (shorter latency is for pleural effusion)***

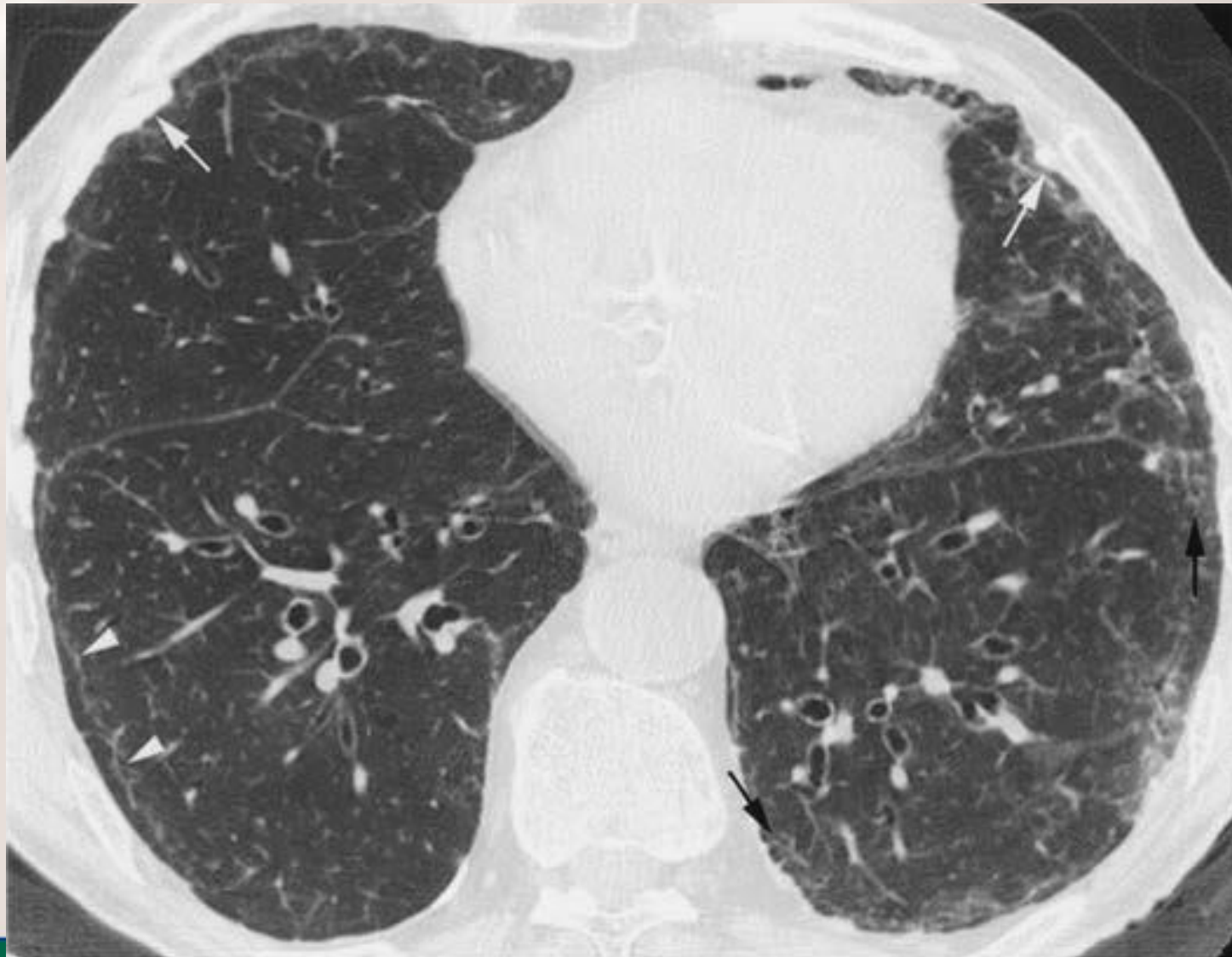
Diagnosis of asbestosis: *CXR*

Pleural plaques



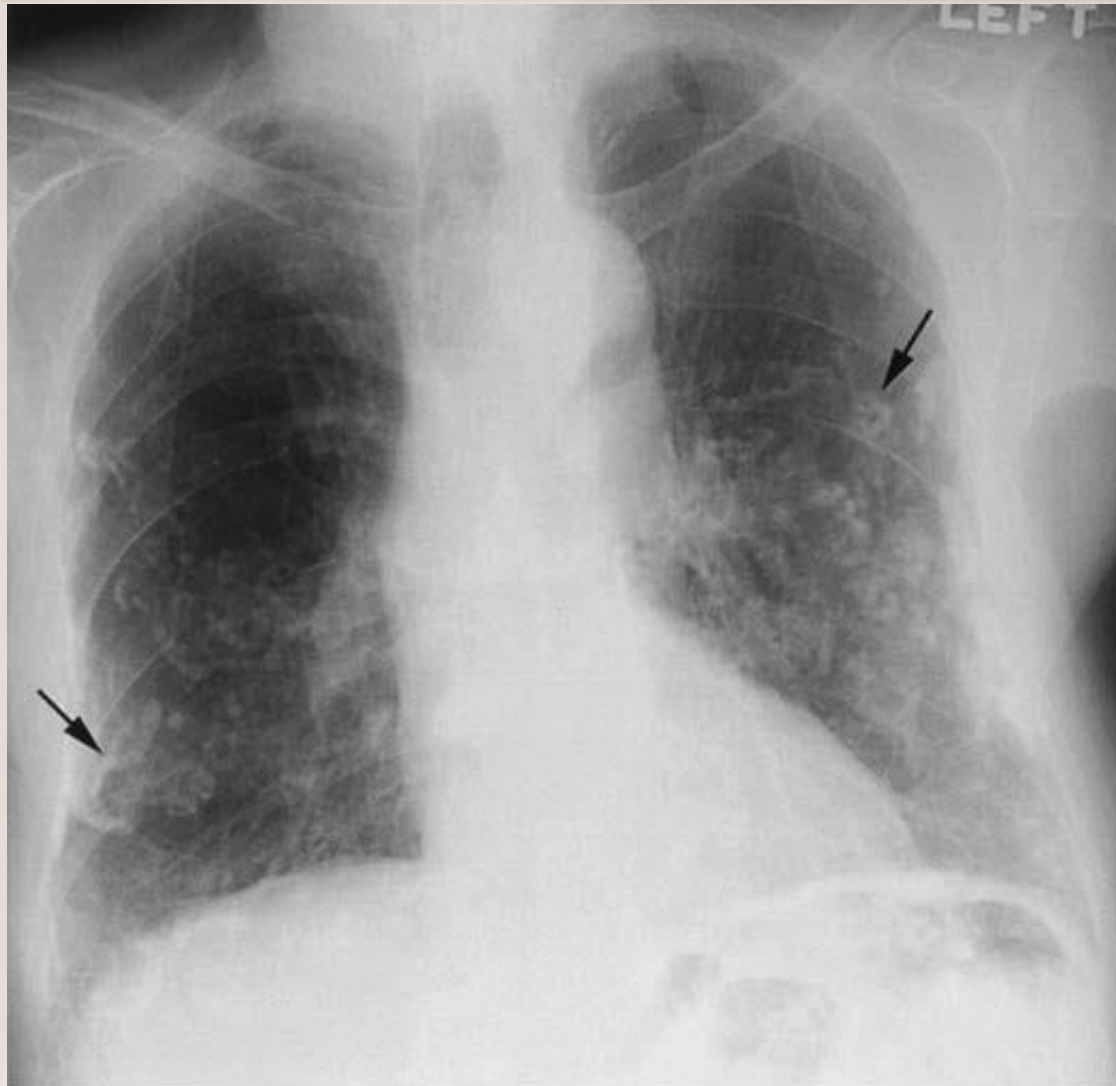
PA radiograph show typical calcified pleural plaques (A) that are essentially diagnostic of asbestos exposure. HRCT scan demonstrates the calcified plaques (B) more clearly but does not add significant diagnostic information.

Asbestosis



B

Asbestosis



A

Prevention and treatment

-
- ❑ *Diseases caused by asbestos inhalation can be prevented by minimizing asbestos dust and fibers in the workplace.*
 - ❑ *Because industries that use asbestos have improved dust control, fewer people develop asbestosis today,*
 - ❑ *but mesotheliomas are still occurring in people who were exposed as many as 40 years ago.*
-

Malignant Pleural Mesothelioma

- ***Tumor arises from the thin serosal membrane surrounding the lungs***
- ***Rapidly invasive***
- ***Rare, although incidences are increasing***
- ***Long latency period: Usually 30-40 years***

Malignant Pleural Mesothelioma

- ***shortterm asbestos exposures of $\leq 1-2$ years, occurring up to 40 years in the past***
- ***>80% of mesotheliomas may be associated with asbestos exposure***


Clinical Presentation

Disease	Signs and Symptoms
<i>Parenchymal Asbestosis</i>	<ul style="list-style-type: none">• <i>Insidious onset of dyspnea on exertion</i>• <i>Fatigue</i>
<i>Asbestos-Related Pleural Abnormalities</i>	<ul style="list-style-type: none">• <i>Usually: None</i>• <i>Sometimes: Progressive dyspnea and intermittent chest pain (depending on the type of pleural abnormality)</i>
<i>Lung Cancer</i>	<ul style="list-style-type: none">• <i>Usually: None (until later stages)</i>• <i>Sometimes: Fatigue, weight loss, or chest pain</i>
<i>Mesothelioma</i>	<ul style="list-style-type: none">• <i>Usually: None (until later stages)</i>• <i>Sometimes: Dyspnea, chest pain, and fatigue</i>

Physical Examination


- ***Focus on lungs, heart, digits, and extremities***
- ***Pulmonary auscultation to detect bibasilar inspiratory rales (not always present)***
- ***Observation of other signs, such as clubbing of the fingers and cyanosis***

CHRONIC EXPOSURE TO ASBESTOS

- inhaled asbestos fibers  deposited in the lungs
- small fibers may be phagocytosed and cleared through lymphatics
- longer fibers are often retained
- Asbestos exposure
 - pleural disease characterized by pleural plaques, effusion, and fibrosis
 - not necessarily affect the lung parenchyma
- asbestosis, with interstitial lung fibrosis resulting from asbestos exposure

ASBESTOSIS


- gradual onset of dyspnea
- risk and severity → extent and duration of exposure
- reaction to retained asbestos fibers in the lung
- similar to those for restrictive lung diseases such as IPF

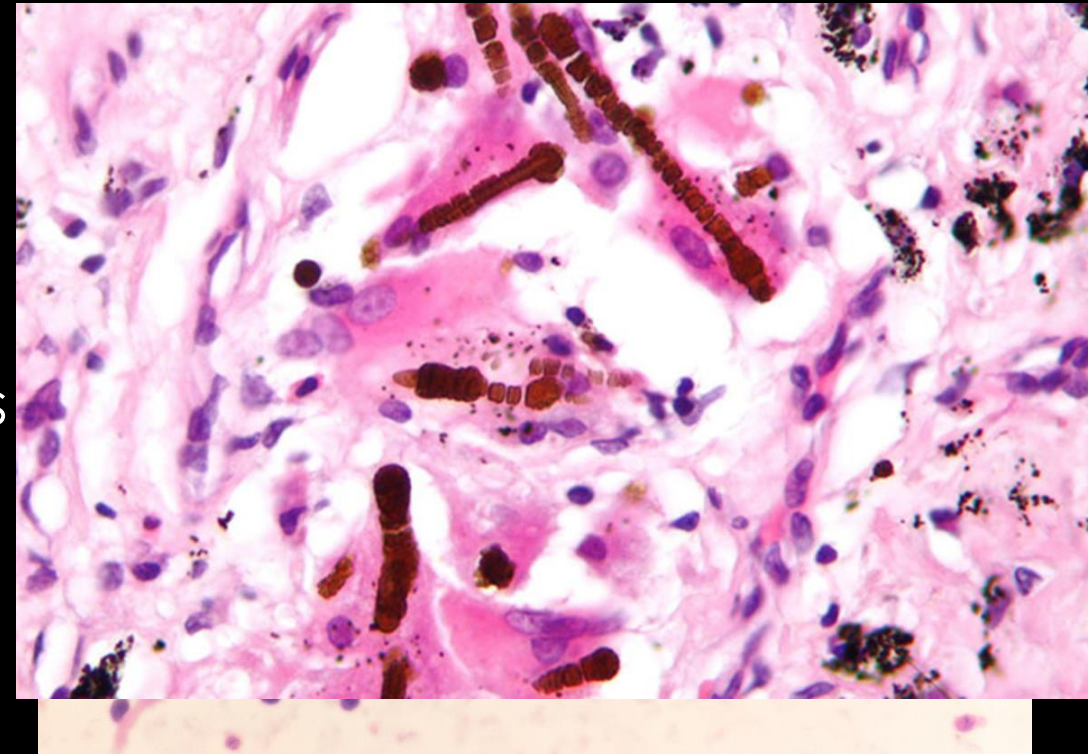


distinguishing
from other
ILDs

- ***detection of significant pleural disease***

DIAGNOSIS

- history of exposure
- concomitant pleural plaques
- lower lobe predominant fibrotic changes
- Tissue specimens  Asbestos bodies





DIAGNOSIS

- Asbestos exposure increases the incidence of malignancy
 - lung carcinoma
 - Mesothelioma
- No specific treatment for asbestosis exists

BERYLLIOSIS

- exposure to beryllium
- rare metal useful in modern, high-technology industries
- acute chemical bronchitis and pneumonitis
- chronic beryllium disease
 - multisystemic granulomatosis that is difficult to distinguish from **sarcoidosis**

DISTINGUISH FROM SARCOIDOSIS

Clinical Finding	CBD	Sarcoidosis
Beryllium lymphocyte proliferation test	Abnormal	Normal
Ophthalmologic	Conjunctivitis only	Conjunctivitis, uveitis, retinal involvement
Erythema nodosum	No	Yes
Lupus pernio	No	Yes
Onset	Insidious	Acute or insidious
Neurologic involvement	None	Can involve the central or peripheral nervous system
Cardiac involvement	Rare	Occasional
Hepatic involvement	Occasional	Common
Isolated hilar adenopathy	Very rare	Common
Extrapulmonary manifestations without pulmonary involvement	No	Yes

Definition of abbreviation: CBD = chronic beryllium disease.



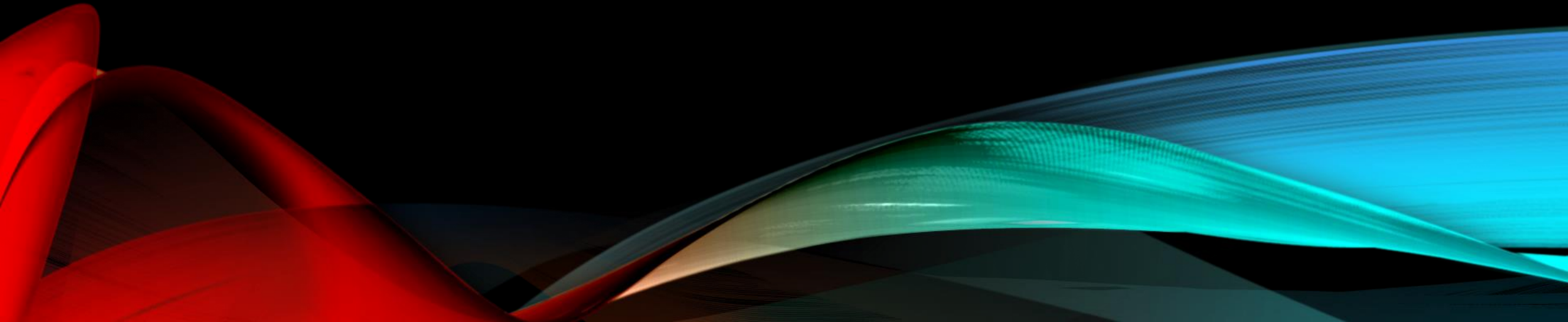
DIAGNOSIS

- history of exposure
- histologic examination
- laboratory confirmation

- beryllium lymphocyte proliferation

- Corticosteroids
- avoid further exposure to beryllium

HYPERSENSITIVITY PNEUMONITIS



DEFNITION AND EPIDEMIOLOGY

- HP (formerly called extrinsic allergic alveolitis)
- common ILD
- exaggerated immune reaction in the alveoli and small airways



- various small, inhaled organic antigens in sensitized individuals

HP

- Potential sensitizing antigens are diverse
- bacterial, fungal, and animal proteins to low-molecular-weight chemicals
- occupational forms of this disease (e.g., paprika splitter's lung)
- More exposures may occur in everyday life
 - hot tub water or to antigens from pet birds →

Farmer's Lung
exposure to moldy hay
thermophilic actinomycetes



PATHOLOGY

- ***Abnormally exuberant immune response to inhaled antigens***
- organic molecules
- small chemical compounds
 - Isocyanates
- susceptible host
 - unclear
 - genetic and environmental factors (e.g., pesticide exposure)

PATHOLOGY

- Smokers are less likely than nonsmokers to develop HP
 - more severe disease course if HP does occur
- After exposure to an antigen, a susceptible individual



develops

- alveolitis with influx of neutrophils and lymphocytes
- TH1-type immune response then leads to granuloma formation

LUNG BIOPSY

- granulomatous inflammation with poorly formed granulomas
- giant cells
- interstitial chronic inflammation
- bronchiolocentric component, and bronchiolitis
- chronic or end-stage disease, fibrosis occurs
 - UIP or NSIP pattern

CLINICAL PRESENTATION ACUTE HP

- several hours flulike illness featuring
 - Fever
 - Chills
 - Cough
 - Dyspnea
 - Malaise
- lasts for up to 24 hours
- farmer's lung from exposure to thermophilic actinomycetes

CLINICAL PRESENTATION SUBACUTE AND CHRONIC HP

- repeated or prolonged lower-level antigen exposure



- chronic dyspnea and cough
- progression to pulmonary fibrosis



- pigeon breeder's lung

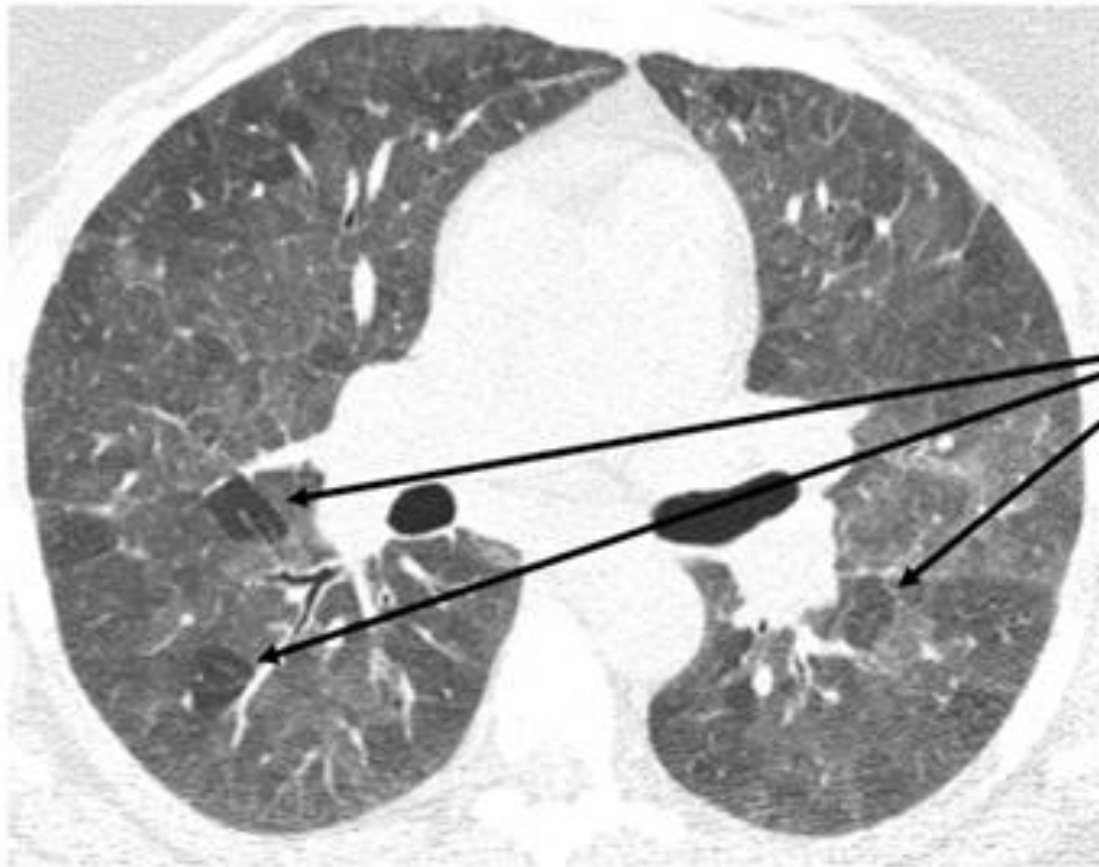
HP

- Acute HP
 - Febrile
 - Diffuse wheezes
- Chronic HP
 - Crackles
 - Clubbing
 - Hypoxemia with exertion
 - hypoxemia at rest in chronic fibrotic HP
 - PFT usually show a restrictive pattern
 - abnormal gas exchange
 - obstructive or mixed patterns

HP

- CXR: nonspecific infiltrates in the middle and upper lung fields
 - normal in acute disease
- CT scanning in acute disease
 - ground-glass opacities
 - centrilobular nodules
 - mosaic attenuation and air trapping patterns resulting from airway obstruction
- Chronic HP
 - architectural distortion with traction bronchiectasis and honeycombing

Hypersensitivity Pneumonitis

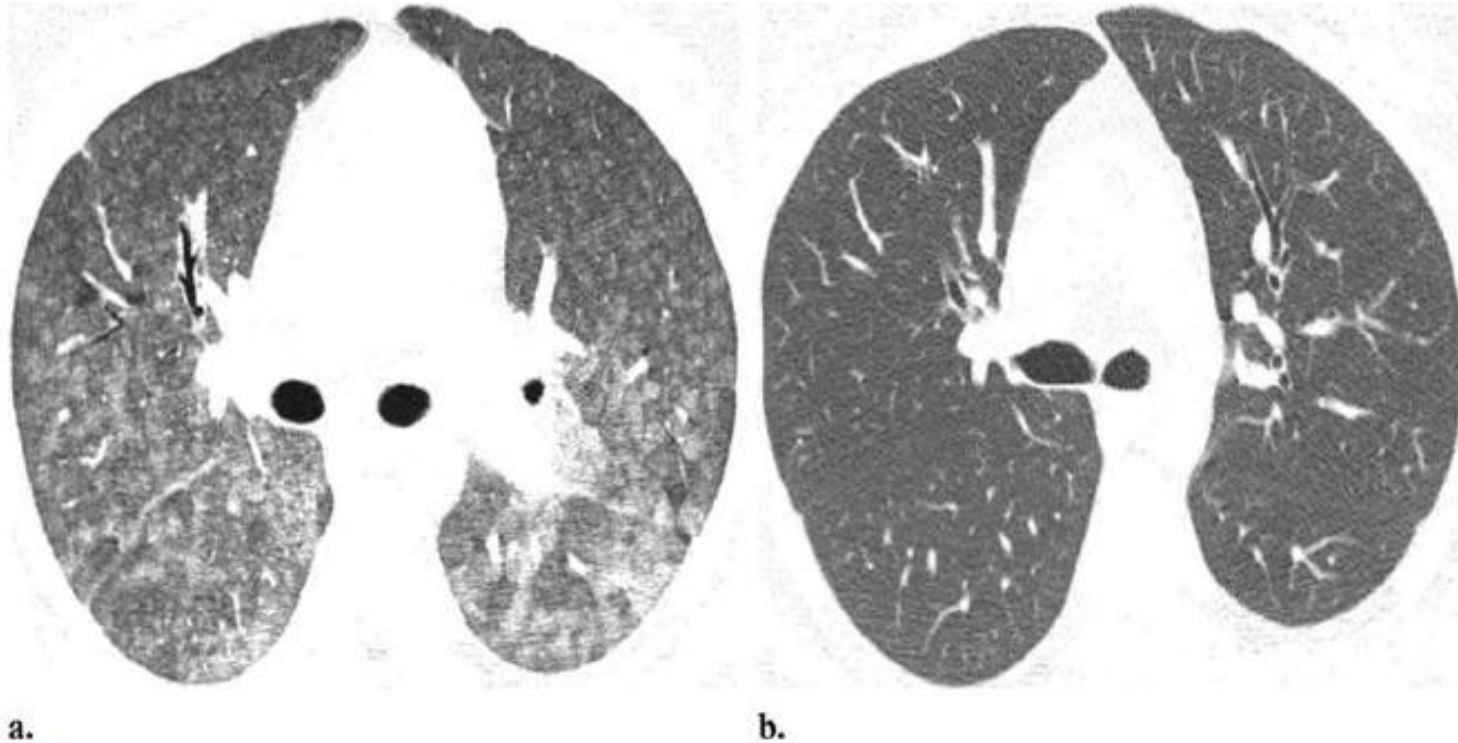


- Mosaic
 - Ground-glass
 - Air trapping

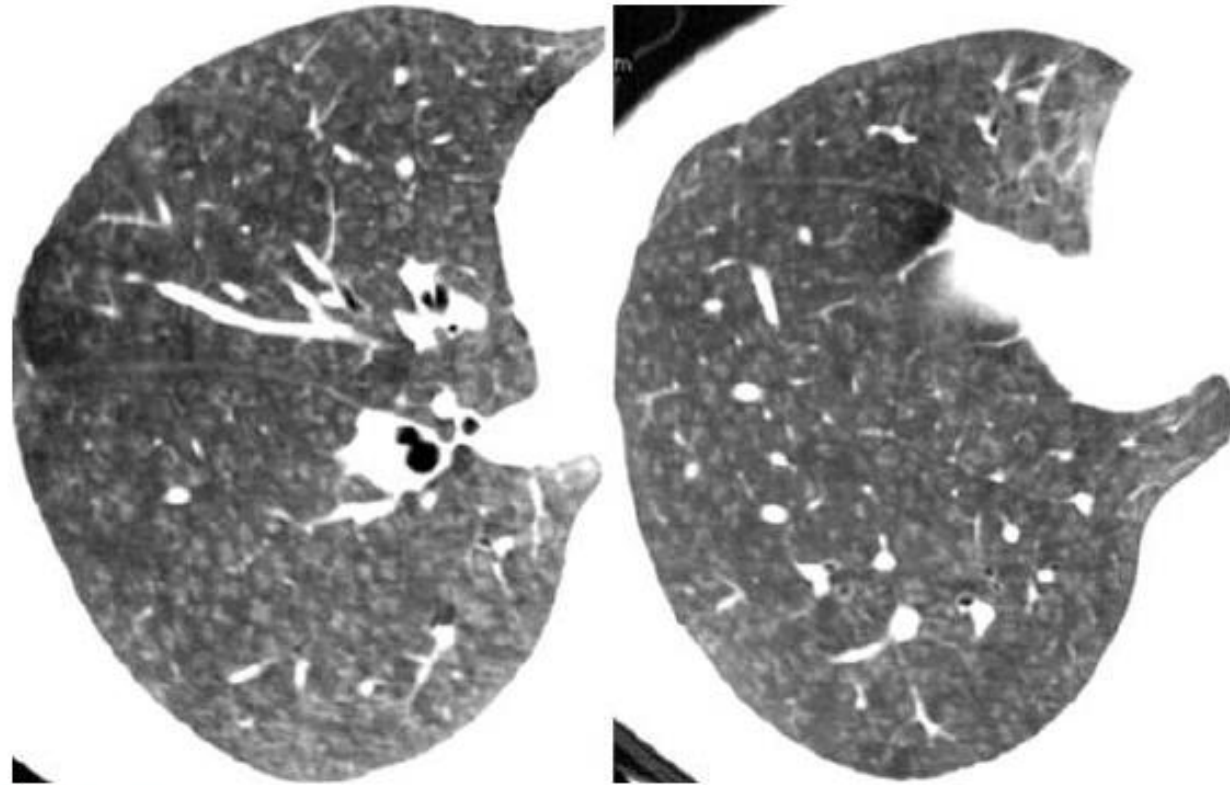
Note: Chronic fibrosis can mimic UIP, but it is usually patchy and less subpleural and lower lung in distribution.

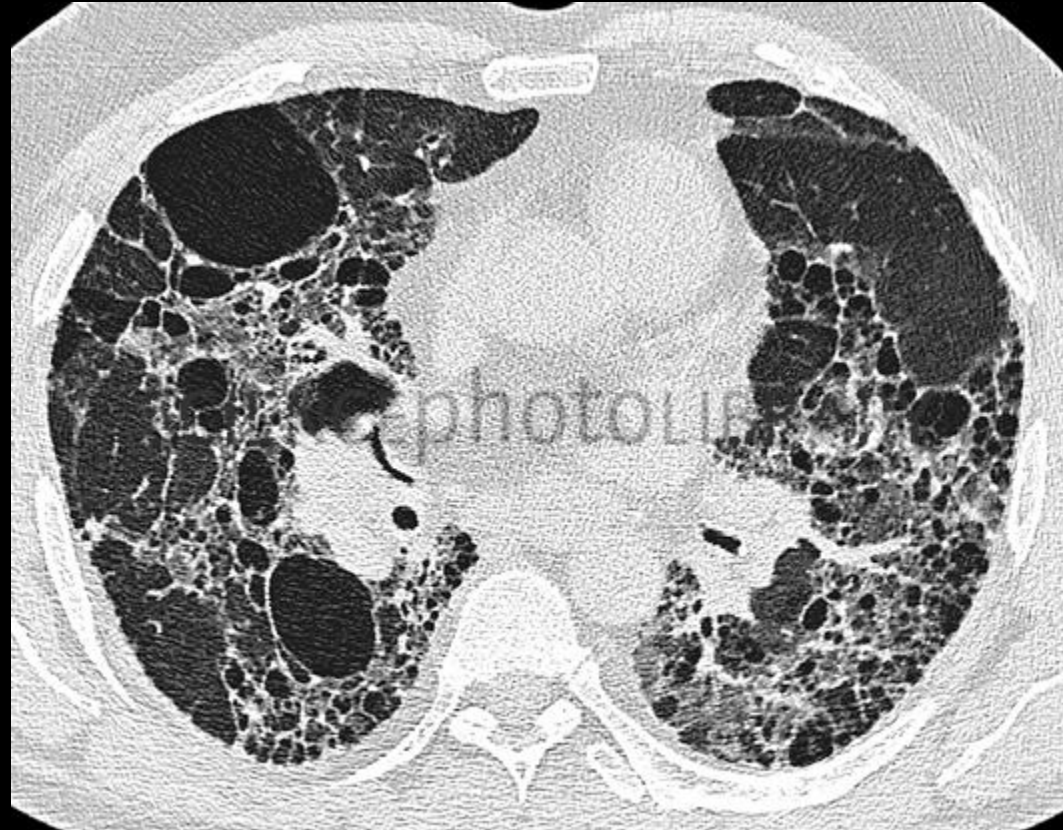
Image courtesy of Kevin R. Flaherty, MD.

Insidious hypersensitivity pneumonitis in a 39-year-old woman with history of exposure to parakeets and cockatiels. (a) HRCT demonstrates extensive ground-glass opacity with a centrilobular concentration. (b) Axial CT image obtained after therapy and removal from exposure shows complete resolution



Insidious hypersensitivity pneumonitis. Axial high-resolution CT images depict ill-defined centrilobular ground-glass opacities





HP

- Emphysema occurs in some cases of advanced farmer's lung.
- BAL
 - lymphocytic alveolitis, with CD4+ T-lymphocyte predominance
- precipitating antibodies to the offending antigen
 - not sufficiently sensitive or specific for diagnosis
 - specific antigen?

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

- ***appropriate exposure, clinical history, BAL, and HRCT imaging***
- lung biopsy
 - necessary for confirmation, especially in subacute and chronic HP
- Transbronchial biopsy
- surgical lung biopsy

Type of Hypersensitivity Pneumonitis	Signs and Symptoms								
	Flu-like Illness (fever, chills, muscle or joint pain, headache)	Rales	Cough	Chronic Bronchitis	Shortness of Breath	Anorexia or Weight Loss	Fatigue	Lung Fibrosis	Clubbing of Fingers or Toes
Acute	✓	✓	✓						
Subacute		✓	✓	✓	✓	✓			
Chronic			✓	✓	✓	✓	✓	✓	✓

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

- ***differential diagnosis***

- Acute HP
 - Acute viral infection
- Chronic HP
 - IPF
 - NSIP
 - Sarcoidosis

TREATMENT

- Clinical improvement often occurs in the hospital setting
 - relapse may occur after discharge
- Corticosteroids can relieve symptoms in the acute phase
 - long-term efficacy in chronic forms?
- Identification of the cause of HP
 - avoidance of exposure to the antigen in chronic hp
- financially or psychologically challenging
 - occupational, pet, or residential exposures
- For advanced HP with fibrosis, lung transplantation



PROGNOSIS

- Acute HP usually has a good prognosis
- Chronic HP can lead to end-stage pulmonary fibrosis and death

